A Dissertation on

endoscopic analysis and survey of the upper gastrointestinal tract

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CERTIFICATE

This is to certify that this dissertation in **"ENDOSCOPIC ANALYSIS AND SURVEY OF THE UPPER GASTROINTESTINAL TRACT"** was a work done by **Dr.K.BARANEE DHARAN** under my guidance during the academic year 2004-2007. This has been submitted in partial fulfillment of the award of M.D.Degree in General Medicine (Branch-I) by the Tamil Nadu Dr.M.G.R. Medical University, Chennai - 600 032.

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DECLARATION

I solemnly declare that this dissertation entitled **"ENDOSCOPIC ANALYSIS AND SURVEY OF THE UPPER GASTROINTESTINAL TRACT"** was done by me at Kilpauk Medical College, during the academic year 2004-2007 under the guidance and supervision of **Prof.Jeevan Kumar, M.D.D.M.**DepartmentofMedical Gasteroenterology & Prof.S.R.Sakunthala,M.D. Depatment of General Medicine This dissertation is submitted to the Tamil Nadu Dr.M.G.R. Medical University, towards the partial fulfillment of requirement for the award of M.D. Degree in General Medicine (Branch - I).

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INTRODUCTION

Dyspepsia affects more than one-fourths of the general population in all industrialised countries and is a frequent reason for medical consultation.

Dysspepsia accounts for upto 7% of hospital visits and 40-70% of gastrointestinal complaints in general medical practice. Dyspepsia appears to have a significant impact upon quality of life.

The definition and management of dyspepsia have underwent a world of change with the advent of endoscopy.

Endoscopy has opened new visitors, for treatment of upper gastrointestinal diseases, be it gastritis or malignancy.

They have given as new options for conservative treatment where previously surgery or just re-assurance were the main stay.

Notwithstanding just visualising the upper GI tract, biopsy and definitive approach for better goal oriented treatment, have become a possibility since 2 decades thanks to upper Gastrointestinal endoscopy.

Even in a terminally ill patients with a carcinoma esophagus an obstructive lesion, can be stented towards palliative means.

But still inspite of the vast strides being made like the Endo USG, there are certain practical set backs in ground reality, which have to be assessed and addressed on a patient to patient basis.

Endoscopy requires costly equipment, technical capability and a willing patient who can afford the procedure.

In the absence of one or any of these, a needy patient should not be denied a meaningful evaluation of his symptoms; Hence this effort was carried out to correlate symptoms and UGI findings on endoscopy.

The present study was conducted to identify the UGI symptoms and determine its role in predicting endoscopic findings.

REVIEW OF LITERATURE

MILESTONES IN ENDOSCOPY

INTRODUCTION

*The earliest exploration of the interior of the human body was attempted in antiquity by Greek, Roman and Arab physicians peering into its orifices through specula, the dim illumination of candle or oil lamp being reflected internally with mirrors.

* Rectoscopes were familiar to Hippocrates (460–377 BC).

* Quite advanced three-and four-pronged dilatation specula were recovered from the ruins of Pompeii (AD 79).

* Marasaumel in the Babylonian Talmud (AD 257) described vaginal specula [4].

THE ERA OF ENDOSCOPY WITH RIGID INSTRUMENTS

The earliest advances were made by urologists, perhaps because the female urethra is one of the shortest conduits into an interior viscus. On a tombstone in a Frankfurt cemetery an epitaph records:

. . . in memory of the devout deceased soul of Philipp Bozzini, medical doctor,German born. This urologist was the first who tried seriously to look into the hollow cavities of the human body by ingeniously conducted light . . .

Philipp Bozzini was born in 1773, aged 35 years, published his experience with the ***Lichtleiter*** (light conductor) which he designed to accommodate different sizes and shapes of specula for the various bodily orifices. The essential components of the instrument were a beeswax candle

as light source and a silver mirror to reflect the light through the speculum .This endeavour marks the beginning of the era of rigid endoscopes [4].

In Paris in 1826 a speculum ***'urethro-cystique'*** was demonstrated by **Pierre Salomon Segalas** to the members of the Academie des Sciences. This was modified from the Bozzini instrument and enabled Segalas to diagnose disorders of the urethra and bladder. It had a safety feature in a gum elastic catheter as introducer [4].

A year later, in the Boston Medical and Surgical Journal, an American, **John Fisher**, published an account of an instrument 'involving the same principles as Segalas' which he claimed to have devised while still a student in 1824.

Fisher suggested Professor Patterson that galvanism might supply an answer to improved illumination of body cavities, a thought that preceded the actual introduction of electricity to endoscopy by some 50 years.

A generation later, in 1853, another urologist in Paris, **Antonin Desormeaux**, rejecting the available electricity- storing batteries as too heavy to move around, introduced the use of a lamp burning a mixture of alcohol and turpentine. To this a series of endoscopic tubes, of various diameters to suit the different orifices, were fitted. His rectoscope, demonstrated to the Academie des Sciences, was 12 cm long and thereafter sigmoidoscopes of increasing length were manufactured. By around 1890 the length of the rigid steel tube stabilised at 30 cm. This remained standard for the sigmoid colon for 60 years until the advent of flexible fibreoptic models [4] .

THE DEVELOPMENT OF ALIMENTARY ENDOSCOPY

The shift of interest to the alimentary tract began in 1868 at a meeting of the Freiburg Society of Naturalists when **Adolf Kussmaul** – with the good sense to use a professional sword-swallower for the demonstration – passed down the oesophagus into his subject's stomach a hollow, rigid metal tube –

the first gastroscope. Illumination was provided by a Desormeaux lamp attached proximally, but visibility was poor.

In 1870 **Joseph Leiter**, a Viennese instrument maker worked on the development of cytoscopes with the urologist **Maximillian Nitze**. A number of others had been experimenting with loops of platinum wire as filaments for electric lamps, the current provided by galvanic batteries. Leiter and Nitze had some success when they devised a method of cooling the lamp and, in 1879, followed up a successful cytoscope with a crude gastroscope using the same technique.

Leiter during his work with **von Mikulicz**, shifted the light to the distal end of the tube but retained Kussmaul's technique for the introduction of his gastroscope .

The prototype oesophagoscopes and gastroscopes were moderately effective but general anaesthesia was required for most subjects.

Fortuitously, in 1885, the International Exhibition of Electricity was held in Vienna. There Leiter saw **Edison**'**s** incandescent electric lamps and promptly adapted the endoscopes to incorporate them.

At the end of the nineteenth century, **Chevalier Jackson**, a prominent American exponent of broncho-scopy, went on to develop, and successfully use, open- tube rigid oesophagoscopes and gastroscopes, under ether-induced general anaesthesia.

In 1896 **Theodore Rosenheim**, in Berlin, published his experience with a triple-tube gastroscope, the innermost tube bearing a row of short-focus lenses, the middle one a lighting system which had reverted to the use of a water-cooled platinum wire loop lamp and an outer tube with a scale of measurement. With existing instruments there remained the need to overcome the problem of the 'blind' areas of the stomach.

George Kelling in Dresden, in 1898, devised a gastroscope with a flexible lower segment, the tip of which could be angulated with a rather clumsy system of wires controlled proximally; this instrument did not find wide favour.

Elsner reintroduced, in 1911, the Rosenheim instrument modified with the safety device of a rubber tip for introduction. Despite the lens system being easily obscured by mucus and gastric contents, it was well taken-up and remained the standard gastroscope for the next 20 years.

Meanwhile, in Munich, **Michael Hoffman**, an optical engineer, had shown that light, and an image, could be conducted around a bend with a flexible tube containing a row of prisms and lenses. This work may have facilitated the next important step in the evolution of gastroscopes [4] .

THE ERA OF SEMI-RIGID ENDOSCOPES

In 1920, in Munich, a charismatic physician, **Rudolf Schindler** improved old Elsner instrument with a facility for insufflating air which largely overcame the problem of the lens smearing. It had the rubber tip mounted separately on an inner tube used on introduction and then withdrawn, and in its place a tube carrying the lens and light system was inserted.

Schindler successfully used the modified Elsner gastroscope until 1932 when he reported his experience with a semi-flexible successor in which the lower third was replaced by a flexible bronze spiral covered in rubber, resulting from a collaboration with the renowned Berlin instrument maker **George Wolf** (1873–1938). Together they devised an inner tube filled with short-focus lenses which could be bent in any direction to an angle of 34º without visual distortion.

The use of this instrument in Rudolf Schindler's uniquely capable hands spelled the end of the era of rigid endoscopy and the semi-flexible successor remained dominant until 1957. His monographs on gastroscopy

and gastric mucosal pathology were groundbreaking and were read extensively in Europe and the US. Would-be endoscopists flocked to Munich for training.

In the light of things to come, mention must be made of **Heinrich Lamm**, a medical student in Munich who, after hearing a lecture by Schindler in 1928, approached him and suggested that a bundle of glass rods might conduct light and images better than a system of lenses. This was a clue relevant to endoscopy which was ignored by optical engineers, physicists and instrument makers for over a century.

Later Schindler was given an appointment in Walter Palmer's department in Chicago as a visiting professor and from this time, Chicago became the new Mecca of endoscopy and a by-product of Schindler's immigration was the promotion in the US of serious interest in the manufacture of endoscopes.

In 1941, the London surgeon Hermon Taylor had the Genito-Urinary Manufacturing Company devise a gastroscope with a flexible distal portion which, with proximal controls, greatly reduced the areas of the stomach that were difficult to visualise directly. This involved an increase in the diameter of the shaft and elongation of the rigid steel portion. Schindler and others in the US were critical of this endoscope [4].

OESOPHAGOSCOPES: FURTHER MODIFICATIONS

Until 1947, oesophagoscopes remained fundamentally unchanged from those used by **von Mikulicz** in the 1880s, except for the incorporation of an **Edison lamp** for illumination.

Edwin Boros in the US, altered the Jackson instrument (to facilitate introduction) by having the most distal portion of the shaft rendered as a metal spiral coil, similar to that used in the Wolf–Schindler gastroscope; this section was then straightened out with a rod after full insertion.

This instrument was superseded in 1949 by the oesophagoscope manufactured by the Eder Instrument Company to the design of A Ray Hufford, especially when further improvement was made substituting a magnifying telescopic eyepiece for the previous lens-in-a-tube system. It became the standard instrument of the day.

In London, meanwhile, as a few physicians in the UK began to undertake oesophagoscopy, **Frances Avery Jones** devised an easy-to-pass slim instrument.

The **Genito-Urinary Manufacturing Company** made in 1956 a wide-bore oesophagoscope with a distal flexible section and a proximal lighting system which overcame the disadvantages of the By 1963, however, the principles of flexible fibreoptics were extended to oesophagoscopes and, in these, the excellent vision, the ease of biopsy and the later addition of a balloon dilatation facility eliminated the need for the traditional rigid instruments for almost all situations [4].

THE THIRD ERA: THE APPLICATION OF FIBREOPTICS;

FULLY FLEXIBLE ENDOSCOPES

The birth of fibreoptic endoscopy in1954:

Hopkins, Baird's and Karl Storz were working independently over the idea of coupling the transmission of light using fibres, an extension in effect of the idea of Hoffmann in Munich referred to earlier.

With his postgraduate fellow Kapany, Hopkins researched the optimum way to coat glass fibres of 0·0025-inch diameter and to arrange them in a bundle so that the spatial relationship of each fibre to its neighbour remained unchanged throughout the length of the bundle. Light and image could then be transmitted even if the bundle was bent through 360º. They suggested the principle could replace the lens in endoscopes.

By 1956, Curtiss and Hirschowitz had resolved the problem of eliminating leakage of light through the wall of individual fibres by coating them with a mixture of highly refractive glass core and low refractoriness, melted together.

A year later they had an assembly of a working fibre bundle of adequate length, a light source intense enough for colour photography, a system for applying torque and a waterproof coating overall. Hirschowitz then passed this bundle on himself without medication or surface anaesthesia and, a week later, successfully on a patient Fibreoptic, fully flexible endoscopy was born. At a meeting in 1957 of the American Gastroscopy Society, Hirschowitz successfully demonstrated the prototype.

Three years later, in 1960, ACMI Ltd produced the first commercial gastroscope, a side viewing instrument with a distal incandescent lamp.

In 1962 Robert Kemp, a Liverpool gastroscopist, suggested the introduction of a controllable directional tip which, taken up by ACMI, greatly improved the capability of the gastroscope.

The Olympus Company introduced a lens-based gastro-camera, the film capsule of which lay in the tip of the gastroscope, but it had limited appeal and was soon replaced by effective 35 mm cameras with synchronised flashes which were mounted on the eyepiece [4] .

EXTENSION OF ENDOSCOPY TO THE PANCREAS AND BILIARY SYSTEMS

In 1966 **Willie Watson**, a Glasgow gastroenterologist viewed the Papilla of Vater. Indeed, in 1965, two radiologists**, Keith Rabinov** and **Morris Simon**, had cannulated the pancreatic duct with a tube introduced through the mouth and fluoroscoped into position. In 1968, the ampulla was cannulated per endoscopy.

By 1970, largely due to ideas from **Itaru Oi** and **K Tagaki**, Japanese endoscope manufacturers produced cannulae with four-way tip control which greatly widened the field of investigation. In 1974, **Classen** and **Demling** split the Papilla of Vater with a bowstring wire diathermy enabling the removal of a gallstone from the biliary tract.

In Britain **Peter Cotton**, first at Middlesex Hospital and later at the Dukes and Carolina venues in the US, became the premier exponent, extending the procedures through from endoscopic sphincterotomy and removal of calculi to lithotripsy and the therapy of biliary and pancreatic Malignancy[4] .

THE ERA OF ELECTRONIC DEPENDENT ENDOSCOPY AND THE FUTURE

In 1983 the first endoscope without fibreoptic transmission of the image was produced by **Welch Alleyn Inc.** in New York. At the tip of the instrument was an electronic sensor consisting of a packed grid of photocell receptors which transmitted images electronically to a video processor and then to a television monitor.

Improved versions became available from the **Olympus Company** and other **Japanese manufacturers**. Subsequently, linkage with a computer enabled automated acquisition of data.

Major changes in practice included the extension of the diagnostic and therapeutic capabilities of endoscopists to the pancreatic and biliary systems and the use of per-ampullary probes, balloons, retrieval baskets for stones and stents for strictures and screening malignancy in situ. The need for intra-abdominal surgery was further restricted.

A major advance, is the incorporation of ultrasound technology. The use of ultrasonic pulses for measuring biological changes in tissues was promoted 50 years ago by **JJ Wild.**

A series of studies of ultrasonic probes attached to endoscopes, later with miniaturised probes passed through the endoscope channels, has imaged lesions in the pancreas, mediastinum and in the peri-gastric and peri-oesophageal tissues, thus facilitating enormously the evaluation of suspect malignancies, operability and also the variceal effects of portal hypertension [4].

Applications in the use of gastrointestinal endoscopy have continued to expand. Indeed a recent survey sponsored by British Society of Gastroenterology suggested that in near future, as many as 1% of the population will undergo upper gastrointestinal endoscopy annually [6].

INSTRUMENTATION

Flexible endoscopic systems transmit light down the endoscope shaft, illuminating the surface to be examined.the image is reflected back and transmitted to the endoscopist either fiberoptically or electronically. In fiberoptic systems, a fixed lens at the end of the instrument shaft focuses the reflected image on an internal fiberoptic bundle. Fiberoptic bundles are 2 to 3 mm wide and are composed of 20,000 to 40,000 individual glass fibers, each approximately 10µm in diameter. Each fiber is coated with a glass of low optical density, which prevents light from escaping. The scope head includes an eyepiece with an adjustable lens; dials which control lens tip deflection; buttons controlling air, water, and suction; and the therapeutic channel inlet [7].

Most modern systems utilize electronic image transmission . These video endoscopes are very similar to the fiberoscopes, offering similar depth of focus, field of view, and tip deflection. The image is reflected on a charge-coupled device (**CCD**) chip mounted on the shaft end. The chip's image contains 30,000 to 150,000 pixels; resolution improves with increasing pixel numbers.The electronic image is transmitted through wires to a video processor in the instrument head [7].

There are two types of colour CCD chips. The earliest devices utilized a mosaic chip, which contains extra pixelsand allows primary-colour filters to be overlaid on the black and white image. These chips can be used with standard xenon light sources. Newer colour video endoscopes use sequential chips, in which all pixels are sequentially illuminated with the light of the three primary colours, alternating each colour 20 to 30 times per minute. Each coloured image is stored transiently in the image processor before being fed to the electron guns in the television monitor. Sequential chips are smaller and can easily mounted on smaller diameter endoscopes. Although they offer better resolution, sequential chips require larger, more expensive light source/ processor units [8].

DEFINITION OF GASTROINTESTINAL (GI) ENDOSCOPIC PROCEDURES

Esophagogastroduodenoscopy (EGD) affords an excellent view of mucosal surfaces of the esophagus, stomach, and proximal duodenum. Standard diagnostic functions include inspection, biopsy, photography and video recording. Diagnostic observations are made concerning focal benign or malignant lesions, diffuse mucosal changes, luminal obstruction, motility, and extrinsic compression by contiguous structures. The most common therapeutic endoscopic procedures include polypectomy, dilatation of strictures, removal of foreign bodies, gastrostomy, and treatment of gastrointestinal bleeding with injection, banding, coagulation or sclerotherapy.

Endoscopic retrograde cholangiopancreatography (ERCP) employs endoscopy to identify the major and minor papillae. The biliary and pancreatic ductal systems are cannulated and opacified with contrast material to provide diagnostic information. Other diagnostic tools may be used in conjunction with ERCP including brush cytology, biopsy, and endoscopic ultrasound. Therapeutic maneuvers included with ERCP include endoscopic sphincterotomy with or without stent placement and with other ancillary techniques for the treatment of pancreatic and biliary duct disease [5].

GENERAL INDICATIONS [9]

These guidelines are based on a critical review of available information and broad clinical consensus, and are as specific and definitive as possible. Clinical considerations may occasionally justify a course of action at variance with these recommendations.

GI ENDOSCOPY IS GENERALLY INDICATED:

1. If a change in management is probable based on results of endoscopy.
2. After an empiric trial of therapy for a suspected benign digestive disorder has been unsuccessful.
3. As the initial method of evaluation as an alternative to radiographic studies.
4. When a primary therapeutic procedure is contemplated.

GI ENDOSCOPY IS GENERALLY NOT INDICATED:

1. When the results will not contribute to a management choice.
2. For periodic follow-up of healed benign disease unless surveillance of a premalignant condition is warranted.

GI ENDOSCOPY IS GENERALLY CONTRAINDICATED :

1. When the risks to patient health or life are judged to outweigh the most favorable benefits of the procedure.
2. When adequate patient cooperation or consent cannot be obtained.
3. When a perforated viscus is known or suspected.

SPECIFIC INDICATIONS STATEMENTS [9]

1. ESOPHAGOGASTRODUODENOSCOPY (EGD) IS GENERALLY INDICATED FOR EVALUATING

A. Upper abdominal symptoms, which persist despite an appropriate trial of therapy.

B. Upper abdominal symptoms associated with other symptoms or signs suggesting serious disease (e.g., anorexia and weight loss) or in patients over 45 years of age.

C. Dysphagia or odynophagia.

D. Esophageal reflux symptoms, which are persistent or recurrent despite appropriate therapy.

E. Persistent vomiting of unknown cause.

F. Other diseases in which the presence of upper GI pathology might modify other planned management. Examples include, patients who have a history of ulcer or GI bleeding who are scheduled for organ transplantation, long-term anticoagulation or chronic nonsteroidal anti-inflammatory drug therapy for arthritis and those with cancer of the head and neck.

G. Familial adenomatous polyposis syndromes.

H. For confirmation and specific histologic diagnosis of radiologically demonstrated lesions:

1. Suspected neoplastic lesion.
2. Gastric or esophageal ulcer.
3. Upper tract stricture or obstruction.

I. Gastrointestinal bleeding

1. In patients with active or recent bleeding.

2. For presumed chronic blood loss and for iron deficiency anemia when the clinical situation suggests an upper GI source or when colonoscopy is negative.

J. When sampling of tissue or fluid is indicated.

K. In patients with suspected portal hypertension to document or treat esophageal varices.

L. To assess acute injury after caustic ingestion.

M. Treatment of bleeding lesions such as ulcers, tumors, vascular abnormalities (e.g., electrocoagulation, heater probe, laser photocoagulation or injection therapy).

N. Banding or sclerotherapy of varices.

O. Removal of foreign bodies.

P. Removal of selected polypoid lesions.

Q. Placement of feeding or drainage tubes (peroral, percutaneous endoscopic gastrostomy, percutaneous endoscopic jejunostomy).

R. Dilation of stenotic lesions (e.g., with transendoscopic balloon dilators or dilation systems employing guidewires).

S. Management of achalasia (e.g., botulinum toxin, balloon dilation).

T. Palliative treatment of stenosing neoplasms (e.g., laser, multipolar electrocoagulation, stent placement) are absent or respond adequately to ulcer therapy.

2. EGD IS GENERALLY NOT INDICATED FOR EVALUATING:

A. Symptoms which are considered functional in origin (there are exceptions in which an endoscopic examination may be done once to rule out organic disease, especially if symptoms are unresponsive to therapy).

B. Metastatic adenocarcinoma of unknown primary site when the results will not alter management.

C. Radiographic findings of:

1. Asymptomatic or uncomplicated sliding hiatal hernia.
2. Uncomplicated duodenal ulcer which has responded to therapy.
3. Deformed duodenal bulb when symptoms are absent or respond adequately to ulcer therapy.

3. SEQUENTIAL OR PERIODIC EGD MAY BE INDICATED:

Surveillance for malignancy in patients with premalignant conditions (i.e., Barrett's esophagus)

4. SEQUENTIAL OR PERIODIC EGD IS GENERALLY NOT INDICATED FOR:

A. Surveillance for malignancy in patients with gastric atrophy, pernicious anemia, or prior gastric operations for benign disease.

B. Surveillance of healed benign disease such as esophagitis, gastric or duodenal ulcer.

C. Surveillance during repeated dilations of benign strictures unless there is a change in status [9] .

ANTIBIOTIC PROPHYLAXIS FOR GI ENDOSCOPY

GI ENDOSCOPY AND THE RISK OF INFECTIOUS COMPLICATIONS

The role of antibiotic prophylaxis is to reduce the possibility of a significant infectious complication.

Despite the large number of endoscopic procedures performed annually, there are few case reports of bacterial endocarditis seen after the procedure. The reported cases of endocarditis were associated with procedures at high risk for bacteremia, such as esophageal dilation, esophageal sclerotherapy and gastroscopy .Other rarely reported infectious complications associated with esophageal sclerotherapy and dilations have included bacterial peritonitis, central nervous system (CNS) infections, and a perinephric abscess. High-risk procedures are those procedures associated with a high incidence of bacteremia [10].

The highest bacteremia rates have been seen in patients undergoing esophageal dilation of a stricture and in sclerotherapy of esophageal varices[10].

CONSENSUS STATEMENTS FOR ANTIBIOTIC PROPHYLAXIS DURING GI ENDOSCOPIC PROCEDURES: Recommendations.

Prophylaxis against infective endocarditis : Regimens

A. Standard general prophylaxis:

amoxicillin 2.0 g by mouth (adult) or 50 mg/kg by mouth (child), 1 hour before the procedure. Alternative for those unable to take by mouth is ampicillin 2.0 g IV/IM (adult) or 50 mg/kg IV/IM (child), within 30 minutes before procedure.

B. Penicillin-allergic patients:

clindamycin 600 mg by mouth (adult) or 20 mg/kg by mouth (child),1 hour before procedure. Alternatives: cephalexin or cefadroxil 2.0 g by mouth (adult) or 50 mg/kg by mouth (child), 1 hour before the procedure; azithromycin or clarithromycin 500 mg by mouth (adult) or 15 mg/kg by mouth (child), 1 hour before the procedure.

C. Penicillin-allergic patients unable to take by mouth:

clindamycin 600 mg IV (adult) or 20 mg/kg IV (child), within 30 minutes before the procedure. Alternative: cefazolin 1.0 g IV/IM (adult) or 25 mg/kg IV/IM (child) within 30 minutes before the procedure; vancomycin 1.0 g IV (adult) or 10-20 mg/kg (child) [10] .

The patient with biliary obstruction, pancreatic pseudocyst, or pancreatic cystic lesion requiring FNA : Recommendation

All patients undergoing ERCP for known or suspected biliary obstruction or known pancreatic pseudocyst should receive antibiotics along with adequate drainage of the biliary obstruction or cyst. Endoscopic transmural drainage of pancreatic pseudocysts, similarly,may result in the introduction of infection into the cystic cavity. In addition, the EUS-guided aspiration of pancreatic cystic lesions also may result in introduction of infection.

Although not supported by randomized, controlled trials, the use of prophylactic antibiotics before attempted drainage of such pseudocysts and similar pancreatic lesions is recommended. Antibiotics that cover biliary flora such as enteric gram-negative organisms, enterococci, and possibly *Pseudomonas sp*. are recommended. Prophylactic antibiotics do not appear to be necessary before FNA of solid masses [10].

RECOMMENDATIONS

- Antibiotic prophylaxis against infective endocarditis is recommended when a high-risk patient is undergoing an endoscopic procedure associated with a high incidence for transient bacteremia.

- Patients undergoing high-risk endoscopic procedures who have a synthetic vascular graft less than 1 year old also should receive antibiotic prophylaxis.

- There is no clear benefit or consensus in the use of prophylactic antibiotics in patients with a prosthetic joint or an orthopedic prosthesis undergoing any endoscopic procedure.

- All patients undergoing ERCP for known or suspected biliary obstruction or known pancreatic pseudocyst should receive antibiotics with adequate drainage of the biliary obstruction or cyst.

- Prophylactic antibiotics are recommended for EUS-guided aspiration of pancreatic cystic lesions but not before FNA of solid masses.

- All patients undergoing endoscopic placement of a percutaneous feeding tube should receive prophylactic antibiotics to limit the risk of soft-tissue infection.

- All patients with cirrhosis who present with GI bleeding should receive prophylactic antibiotics to decrease infectious complications and mortality [10].

PREPARATION OF PATIENTS FOR GI ENDOSCOPY

INTERVENTIONS AND PRACTICES CONSIDERED

General

1. Perform preprocedure assessment of patient and review of medical records, including history of medical illnesses, medications, past surgery, previous endoscopies, and history of drug allergies or bleeding tendencies

2. Obtain and record informed consent.

3. Provide discussion of what will be done, expected discomfort, potential risks and benefits of the procedure including those of sedation, alternative methods of investigation or management.

4. Provide instructions to restrict activities requiring alertness (e.g., driving, operating heavy or potentially harmful machinery, making legally binding decisions) until the effects of the medications are completely gone.

5. Review instructions before procedure, and provide written instructions, including steps to follow in the event of a complication, upon discharge[11].

Upper Gastrointestinal (GI) Endoscopy

1. Preprocedure fasting (no solids for 6 hours, no liquids for 4 hours before procedure)

2. Topical pharyngeal anesthesia including 20% benzocaine spray

3. Anticholinergic agents including atropine (not for routine use)

4. Parenteral glucagons [11].

Endoscopic Ultrasound (EUS)

1. Preparation as for upper GI endoscopy
2. Sedatives
3. Prophylactic antibiotics (for patients requiring fine needle aspiration [FNA] of cystic lesions) [11].

Special Considerations

1. Electronic monitoring of pulse, blood pressure, oxygen saturation, capnography and continuous electrocardiographic (ECG) rhythm
2. Prophylactic antibiotics in patients undergoing certain procedures (e.g., esophageal dilation) in high-risk patients (e.g., prosthetic valve)
3. Measurement of coagulation parameters and adjustments to anticoagulation therapy (e.g., aspirin or other nonsteroidal anti-inflammatory drugs), if necessary
4. Cardiac monitoring during use of electrosurgical equipment in patients with cardiodefibrillators
5. Administration of insulin/hypoglycemic agents in diabetic patients [11]

CONSENT FOR ENDOSCOPY

To protect the patient's right of self-determination, informed consent should be obtained and documented before the patient is medicated. This must include a discussion of what will be done, expected discomfort, potential risks and benefits of the procedure including those of sedation, alternative methods of investigation or management, and the opportunity to ask questions[12 -14]. Appropriate efforts are needed to address specific circumstances resulting in any patient's inability to provide informed consent.

MEDICATION FOR ENDOSCOPY

Medication before and during endoscopic procedures may be used to diminish GI secretions or motility, decrease the patient's anxiety or discomfort, and to provide amnesia [15].

The guiding principle must be patient comfort and safety. General anesthesia or the presence of an anesthesiologist is indicated in special circumstances. The ASGE has recently published guidelines on the use of deep sedation and general anesthesia during endoscopic procedures [16].

Anesthetic agents such as propofol and sedation adjuncts such as droperidol, promethazine, and diphenhydramine are useful in certain patients undergoing endoscopic procedures. While propofol provides faster onset and deeper sedation than standard benzodiazepines and narcotics, as well as faster recovery, clinically important benefits have not been consistently demonstrated in average-risk patients undergoing standard upper and lower endoscopy. The routine use of propofol in these patients cannot currently be endorsed. For prolonged therapeutic procedures, these agents have been demonstrated to be superior to standard benzodiazepine/ narcotic sedation and their use should be considered . Deep sedation requires more intensive monitoring by trained individuals. The assistance of anesthesiologists should be considered in patients undergoing prolonged therapeutic procedures requiring deep sedation, those with anticipated intolerance of standard sedatives, and those at increased risk for sedation-related complications, such as patients with severe comorbidities or with anatomic variants increasing the risk of airway obstruction. The use of agents to achieve sedation for endoscopy must conform to the individual institution's policies [17-19].

Appropriate Personnel and Equipment for Propofol Use in an Endoscopic Procedure Room

- At least one person who is qualified in both basic and advanced life support skills (i.e., tracheal intubation, defibrillation, use of resuscitation medications)
- Physiologic monitoring should include pulse oximetry, electrocardiography, and automated blood pressure measurement. *Monitoring oxygenation by pulse oximetry is not a substitute for monitoring ventilatory function*.
- Equipment for airway management and resuscitation
- Trained personnel dedicated to the *continuous and uninterrupted* monitoring of the patient's physiologic parameters and administration of propofol
- Extended monitoring with capnography should be considered, as it may decrease the risks during deep sedation [17 -19] .

Guideline for Anesthesiology Assistance during Gastrointestinal Endoscopy

Anesthesiologist assistance may be considered in the following situations:

- Prolonged or therapeutic endoscopic procedure requiring deep sedation
- Anticipated intolerance to standard sedatives
- Increased risk for complication due to severe comorbidity (American Society of Anesthesiologists [ASA] class III physical status classification or greater)
- Increased risk for airway obstruction due to anatomic variant

There has been recent interest in the use of **propofol**, a rapidly acting anesthetic that provides excellent sedation and amnesia with a significantly shorter recovery time when compared with sedatives and/or analgesics [17-19].

The choice of sedative is largely operator dependent, but generally consists of benzodiazepines used either alone or in combination with an opiate. The most commonly used benzodiazepines are midazolam and diazepam. The efficacy of sedation with these two benzodiazepines is comparable [20]. However, most endoscopists favor midazolam for its fast onset of action, short duration of action, and high amnestic properties.

PHARYNGEAL ANESTHESIA

Pharyngeal anesthesia is often used to suppress the gag reflex during procedures involving the upper GI tract. Commonly used topical anesthetics include benzocaine, tetracaine, and lidocaine. They are administered by aerosol spray or gargling. The effects last for up to 1 hour. Despite their widespread use, there are conflicting data on their benefit. One study has suggested that topical anesthesia produced no additional benefit when used with intravenous conscious sedation[21, 22]. Another study suggested that the benefit might be greatest for patients who are less than 40 years old, those undergoing the procedure for the first time, or patients who are particularly anxious [23].There are numerous case reports on the occurrence of methemoglobinemia after administration of topical anesthetics. This should be suspected by the presence of clinical “cyanosis” in the face of a normal arterial PO_2 [24].

POSTPROCEDURE MONITORING [25]

After completion of endoscopic procedures, patients are to be observed for adverse effects from either instrumentation or sedation. The length of the follow-up observation is dependent on the perceived risk to the patient. Patients may be discharged from the endoscopy unit or postprocedure recovery area once vital signs are stable and the patient has reached an appropriate level of consciousness. Despite the appearance of appropriate recovery, it is well recognized that patients may have a prolonged period of amnesia and/or impairedjudgment and reflexes after intravenous medications administered to induce sedation.

Patients should be advised before the administration of sedatives that a prolonged period of impaired cognition may occur. They should be instructed to make plans not to drive, operate heavy or potentially harmful machinery, or make legally binding decisions. When sedatives are administered, a competent companion for discharge must accompany patients from the recovery area. Written instructions upon discharge are necessary as the amnestic period following sedation is variable.

Postprocedure instruction on the signs and symptom of potential adverse outcomes and complications is also advisable. Patients should be given written instructions on steps to follow in the event of a complication, including a phone number where 24-hour-a day coverage is available in the event of an emergency [25].

SPECIAL CIRCUMSTANCES

No sedation

Selected patients may be able to undergo endoscopic procedures with no sedation. Ultrathin endoscopes with diameter from 5.3 to 6 mm can improve the tolerability of upper endoscopy and may be used without

sedation[26-28]. In general, topical anesthesia is used. There are several studies demonstrating successful colonoscopy in patients who receive no sedation or sedation only if needed [29-32]. Older patients, men, patients who are not anxious, or patients without a history of abdominal pain may have better tolerance of upper endoscopy or colonoscopy with little or no sedation. For procedures performed without medications, it is still prudent to use varying levels of monitoring as the situation demands.

ROLE OF EGD IN UGI – BLEED

DEFINITION

Upper-GI bleeding refers to GI blood loss whose origin is proximal to the ligament of Treitz. Acute UGIB can manifest as hematemesis, ‘‘coffee ground’’ emesis, the return of red blood via nasogastric tube and/or melena with or without hemodynamic compromise. Hematochezia (bright red blood per rectum) may occur in patients with extremely brisk UGIB [33].

Upper gastrointestinal (UGI) bleeding is a common medical presentation for patients seen by gastroenterologists and is associated with significant morbidity, mortality and the use of healthcare resources [34, 35].

Endoscopy should be considered a primary and pivotal early intervention in establishing the source of bleeding. Early endoscopy allows clinicians an opportunity for therapeutic interventions and estimation of an individual’s risk for recurrent bleeding[34, 35].

TIMING OF ENDOSCOPY

Endoscopy with the intention of therapeutic intervention(s) should be considered strongly as an early intervention to control bleeding and prevent rebleeding. Several issues regarding the exact timing of endoscopy and post endoscopy management are less clear [36].

The timing of endoscopy remains a significant controversy and few studies actually address this issue directly. While intuitively endoscopy with the intent of a therapeutic intervention is expected to improve short-term medical outcomes, the use of early endoscopy is difficult to define. In general, most studies evaluate the use of endoscopy within 24 hours of presentation. However, it remains unclear regarding the most optimal timing within the first 24 hours [36].

Emergent endoscopy is generally performed for patients who cannot be hemodynamically stabilized, those presenting with orthostasis, tachycardia, shock and/or signs of continued bleeding. The rationale is that hemostasis can be achieved with therapeutic endoscopic intervention and provide clinicians an opportunity to stabilize the patient hemodynamically [36].

In contrast, endoscopy may be performed under more controlled conditions (but within 24 hours) and after more complete resuscitation in patients who do not have evidence of continued bleeding and who are medically stable. Multiple studies have demonstrated that therapeutic endoscopy using epinephrine injection, sclerosing agents, electrocautery, heater probes and other hemostatic interventions facilitate early control of bleeding reduce rebleeding rates and improve short-term morbidity and mortality [36].

NON VARICEAL UGI BLEED

ENDOSCOPIC TREATMENT MODALITIES FOR GI HEMORRHAGE

Injection Methods.

* The method of action of injection therapy is primary tamponade because of volume effect, with some agents having a secondary pharmacologic effect. Agents available for injection to produce tamponade include normal saline solution and epinephrine (adrenaline).

* Sclerosants such as ethanol, ethanolamine, and polidocanol are not used to produce tamponade but instead cause direct tissue injury and thrombosis.

* Agents also can be used in combination (such as epinephrine followed by ethanolamine).Limited data suggest that higher volumes of epinephrine injected at endoscopy have a superior effect in achieving hemostasis [37].

* A separate class of injectable agents includes thrombin, fibrin, and cyanoacrylate glues, which are used to create a primary tissue seal at a bleeding site [38].

* Thrombin has been used in several studies in conjunction with heat probe therapy [38] and epinephrine injection [39, 40].

Cautery

* Cautery devices include heat probes neodymium-yttrium aluminum garnet lasers, argon plasma coagulation (APC), and electrocautery probes. Laser therapy is not widely used in many centers because of cost, training, and support issues.

* Electrocautery refers to the use of monopolar electrocauteryor bipolar (multipolar) electrocautery.

* Heat probes and electrocautery probes also use local tamponade (mechanical pressure of the probe tip at/ on the bleeding site) combined with heat /electrical current to coagulate (and thus close) the vessel in question, a process known as coaptation.

* Argon plasma coagulation uses a stream of ionized gas to conduct electricity resulting in coagulation of superficial tissues. Argon plasma coagulation is primarily used for the treatment of superficial lesions, such as vascular abnormalities, but may have a role in some patients with bleeding from other causes [41].

Mechanical Therapy

Mechanical therapy refers to the implantation of a device that causes physical tamponade of a bleeding site. Currently, the only mechanical therapies widely available are endoscopically placed clips and band ligation devices.

Endoscopic clips usually are placed over a bleeding site (e.g., visible vessel) and left in place. Clips currently are available in two or three pronged configurations, can be affixed to bleeding sites, and typically slough off days to weeks after placement [42].

BLEEDING GASTRIC OR DUODENAL ULCERS

Gastric or duodenal ulcers (Figure 1, F) are the most common causes of UGI bleeding and there is a large volume of literature evaluating the prognosis associated with the endoscopic treatment of high risk ulcer lesions [43]**.** At the time of endoscopy and in the presence of gastric or duodenal ulcers, a test for *Helicobacter pylori* should be obtained. When positive, *H.pylori*

eradication has been shown to reduce the long-term (1 year) rate of rebleeding as compared to no treatment [44].

ACTIVELY BLEEDING ULCERS AND ULCERS WITH NON-BLEEDING VISIBLE VESSELS

Actively bleeding ulcers and ulcers with nonbleeding visible vessels are associated with the greatest risk of poor outcomes and / or re-bleeding and are seen collectively in up to 35% of patients with ulcers at the time of endoscopy [45]**.** For this reason, endoscopy should be always performed with the intention for therapeutic intervention. Endoscopic therapies may include injection of epinephrine and / or sclerosants, electrocautery, heater probe or a combination of injection with subsequent thermal therapies. While the optimal choice of therapy is debated, studies repeatedly show that the use of these hemostatic therapies reduce the rate of re-bleeding as compared to no intervention**.**

RECURRENT BLEEDING AFTER ENDOSCOPIC TREATMENT

Despite adequate initial endoscopic therapy, recurrent bleeding in patients with UGIB can occur in up to 24% of high-risk patients, although more recent studies that emphasize the use of PPI therapy in addition to combination endoscopic therapy show recurrent bleeding rates of approximately 10% [46, 47]. Studies suggest that if re-bleeding occurs, it will be within 48-72 hours [48 -50]. .Patients with recurrent bleeding respond favorably to repeat endoscopic therapy**.**

Scheduled repeat endoscopy (e.g., at 24 hours) has been advocated for patients with high-risk stigmata that were treated at the time of the initial bleed [51].

PREDICTORS OF RE- BLEEDING [52]

1. Older age
2. Shock/hemodynamic instability/orthostasis
3. Comorbid disease states (e.g., coronary artery disease, congestive heart failure, renal and hepatic diseases, cancer)
4. Specific endoscopic diagnosis (e.g., GI malignancy)
5. Use of anticoagulants/coagulopathy
6. Presence of a high-risk lesion (e.g., arterial bleeding, nonbleeding, visible vessel and clot)

Recent data has suggested that patients with overlying/ adherent clots may benefit from removal of the clot and therapeutic intervention based on the appearance of the ulcer base [53]. A clot may be removed by simple irrigation to expose the underlying ulcer bases. If the clot dislodges, the ulcer base can be inspected for the presence of a visible vessel or acute bleeding; appropriate action can be taken. If the clot remains adherent, the endoscopist may opt for medical management. Alternatively, if the clot cannot be removed by simple irrigation, a preliminary study reports reduced rate of bleeding with epinephrine injection into the base of the base of the adherent clot, by clot removal and application of thermal contact therapy [54].

ROLE OF EGD IN VASCULAR ABNORMALITIES

Vascular malformations typically cause microscopic chronic blood loss and, occasionally, acute GI hemorrhage. These lesions can occur sporadically or in association with other disorders: cirrhosis, renal failure, radiation injury, various collagen vascular diseases, and hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu disease). Endoscopic ligation [55], laser, APC, contact cautery, and sclerotherapy have been reported to be effective[56].

There are no prospective trials comparing treatment methods for acute UGIB caused by vascular malformations.

Dieulafoy's lesion typically presents with intermittent, recurrent UGIB [57]. Endoscopic methods to treat Dieulafoy's lesion include banding, clipping, electrocautery, cyanoacrylate glue, sclerosant injection, epinephrine injection, heat probe, banding, and laser therapy. Large single-center experiences have not identified one modality as being superior to others, and no prospective randomized trials have been published [58 -60].

ROLE OF EGD IN VARICEAL BLEED

Variceal bleeding is a common and serious complication of portal hypertension. Mortality after the index hemorrhage in patients with cirrhosis has been reported to be as high as 50%, with a 30% mortality associated with subsequent bleeding episodes.

SCREENING FOR ESOPHAGEAL VARICES

Effective prophylactic treatments to prevent variceal bleeding exist for patients with esophageal varices. There no reliable methods of predicting which cirrhotic patients will have esophageal varices without endoscopy.

An American Association for the Study of Liver Diseases guideline suggests that patients with Child's stage A liver cirrhosis and signs of portal hypertension, specifically a platelet count of less than 140,000mm^3 ,and / or enlarged portal vein diameter of greater that 13 mm or those classified as Child's B or C at diagnosis should have screening endoscopy.

Patients with cholestatic disease may have portal hypertension with relatively preserved liver function and platelet counts. A retrospective study of 235 patients concluded that patients with either primary biliary cirrhosis or primary sclerosing cholangitis who have a count <200/mm^3 , an albumin level

<40 gm/L, and a bilirubin level >20 mmol/L should be screened for esophageal varices.

Other groups recommend screening for all patients diagnosed with cirrhosis. The optimal surveillance intervals for esophageal varices have not been determined. For patients found to have no varices on initial screening endoscopy, repeat endoscopy at 3-year intervals has been suggested, whereas patients with small varices should undergo endoscopy in 1 to 2 years.

Esophageal varices may grow faster in patients with cirrhosis secondary to alcohol abuse or severe liver impairment and in those with endoscopic stigmata of high risk (‘‘red wale markings’’); this subgroup of patients should undergo yearly upper endoscopy [61].

PRIMARY PROPHYLAXIS

Endoscopic sclerotherapy (EST) is not recommended for primary prophylaxis. While several studies have shown benefit, a well-done US study showed an increased mortality rate in the treated group.

Endoscopic variceal ligation (EVL) eradicates esophageal varices with fewer complications than ESTand has been shown to be as effective as the use of beta-blockers [61].

ENDOSCOPIC TREATMENTS FOR VARICEAL HEMORRHAGE

ENDOSCOPIC VARICEAL LIGATION

EVL has become the treatment of choice both for controlling variceal hemorrhage and for variceal obliteration in secondary prophylaxis.

A meta-analysis has confirmed the superiority of EVL over EST for all major outcomes (recurrent bleeding, local complications including ulceration and stricture formation, time to variceal obliteration, and survival).

Recurrence of esophageal varices may develop more frequently in those treated with EVL, and regular endoscopic surveillance remains a critical aspect of management.

The introduction of multiple-band firing devices has made EVL more widely acceptable and it is favored by many over EST for eradication of esophageal varices [61].

ENDOSCOPIC SCLEROTHERAPY

EST is successful in controlling active bleeding in over 90% of patients and can reduce the frequency and severity of recurrent variceal hemorrhage. Gastric varices in continuity with esophageal varices may be treated with EST below the esophagogastric junction [61] .

GASTRIC VARICES --- A SPECIAL MENTION

Gastric varices are most commonly located in the cardia in continuity with esophageal varices. Isolated gastric varices are most commonly located in the fundus and can be seen in patients with cirrhosis and portal hypertension, as well as in patients with splenic vein thrombosis (e.g., from

pancreatic disease) or portal vein thrombosis. Bleeding from gastric varices is typically high volume in nature and can present with massive hematemesis. In general, endoscopic therapy for the treatment of bleeding gastric varices has been less successful than for esophageal varices. Treatment options that have been studied in prospective trials include injection of ***cyanoacrylate-based tissue adhesives***, alcohol, sclerosants, and the use of band ligation [61].

ROLE OF EGD IN OBSCURE GI BLEED (OGIB)

Obscure GI bleeding (OGIB) has been defined as bleeding of unknown origin that persists or recurs after an initial negative endoscopic evaluation, including colonoscopy and/or upper endoscopy (EGD) [62].

UPPER ENDOSCOPY

EGD is indicated for the initial evaluation of a suspected upper GI source of bleeding. A repeat examination may yield a source even when the initial EGD was negative. One study suggested that up to 64% of lesions identified with a push enteroscope were within reach of a standard endoscope. The investigators suggested that repeat EGD should be considered before push enteroscopy (PE) for patients with OGIB. Conditions that might increase the yield of repeat EGD include large hiatal hernias and a history of NSAID use. If GI bleeding has not been documented clearly in the presence of iron deficiency anaemia (IDA), one must consider a small bowel biopsy to evaluate for celiac sprue at the time of EGD, although studies are mixed on the yield of small bowel biopsy in IDA [62].

PUSH ENTEROSCOPY

Push enteroscopy (PE), whereby a long endoscope is inserted into the jejunum through the mouth, is used to evaluate a larger segment of the small intestine, particularly in the setting of OGIB. The diagnostic yield is approximately 40% to 65% [62].

CAPSULE ENDOSCOPY

Wireless video capsule endoscopy (CE) is a new technology that enables endoscopic evaluation of the small intestine. This new technology has the potential to identify a source of bleeding in patients with OGIB and/or IDA [62].

SURGERY

Intra-operative enteroscopy. Intra-operative enteroscopy (IOE) during laparotomy is typically used as a last resort in patients with OGIB requiring multiple transfusions and/or repeated hospitalizations [62].

ROLE OF ENDOSCOPY IN THE SURVEILLANCE OF PREMALIGNANT CONDITIONS OF THE UPPER GI TRACT [63]

* Patients with chronic GERD at risk for Barrett's esophagus should be considered for endoscopic screening .

* In patients with Barrett's esophagus without dysplasia, the cost effectiveness of surveillance endoscopy is controversial. If surveillance is performed, an interval of 3 years is acceptable .

* Although an increased cancer risk has not been established in patients with Barrett's esophagus and low grade dysplasia, endoscopy at 6 months and yearly thereafter should be considered.

* Patients with Barrett's esophagus with confirmed HGD should be considered for surgery or aggressive endoscopic therapy. Patients with HGD who elect endoscopic surveillance should be followed-up closely (ie, every 3 months) for at least 1 year. If no further HGD is confirmed, then the interval between follow-ups may be lengthened.

* There are insufficient data to recommend routine surveillance for patients with achalasia.

* Patients with a severe caustic esophageal injury should undergo surveillance every 1 to 3 years beginning 15 to 20 years after the injury.

* Patients with tylosis should undergo surveillance endoscopy every 1 to 3 years beginning at age 30 years.

* There are insufficient data to support routine endoscopic surveillance for patients with previous aerodigestive squamous cell cancer.

* Adenomatous gastric polyps should be resected because of the risk for malignant transformation. Adenomatous polyps may recur in synchronous and metachronous sites, and surveillance endoscopies should be performed at 3- to 5-year intervals.

* Endoscopic surveillance for gastric intestinal metaplasia has not been extensively studied in the U.S. and therefore cannot be routinely recommended. However, there may be a subgroup of high-risk patients who will benefit from endoscopic surveillance .

* Patients with confirmed gastric high-grade dysplasia should be considered for gastrectomy or local resection because of the high incidence of prevalent carcinoma.

* Patients with pernicious anemia may be considered for a single screening endoscopy, particularly if symptomatic, but there are insufficient data to recommend ongoing surveillance.

* There are insufficient data to support routine endoscopic surveillance in patients with previous partial gastrectomy for peptic ulcer disease.

* Patients with FAP should undergo regular surveillance endoscopy using both end-viewing and side-viewing endoscopes, starting around the time of colectomy or after the age of 30 years.

* Patients with HNPCC have an increased risk of gastric and small-bowel cancer. Surveillance should be strongly considered [63].

COMPLICATIONS OF UPPER GI ENDOSCOPY

Major complications related to diagnostic procedures can be broken down into cardiopulmonary complications, complications related to sedation, infectious complications, perforation, and bleeding.

CARDIOPULMONARY COMPLICATIONS/ COMPLICATIONS RELATED TO SEDATION

Cardiopulmonary complications related to sedation and analgesia are the most common type of complication seen with diagnostic endoscopy. These complications range from minor changes in vital signs to myocardial infarctions, respiratory depression, and shock / hypotension.

It is estimated that oxygen desaturation may occur in up to 70% of patients undergoing various endoscopic examinations; more severe desaturation occurs less commonly.

Sedation-related complications are generally identified during the procedure. Appropriate management includes “basic life support” if necessary. Proper management requires the presence of resuscitation medications, including reversal agents and equipment in all areas where endoscopy is performed [64].

INFECTIOUS COMPLICATIONS

Infectious complications related to diagnostic endoscopy result either from the procedure itself or from the use of contaminated equipment.

Transient bacteremia may occur during a diagnostic endoscopic procedureand is found more often for therapeutic procedures.Uncommon complications include retropharyngeal and retroesophageal abscesses in patients who have had difficult intubations. This may be related to retropharyngeal trauma and/or nonclinically apparent perforations should be considered [64].

PERFORATION

Perforation of the upper GI tract related to diagnostic endoscopy is relatively low. Predisposing factors to perforations include the presence of anterior cervical osteophytes, Zenker's diverticulum, esophageal strictures, and malignancies. Although uncommon, perforations of the esophagus are associated with a relatively high mortality rate that approximates 25% [64].

BLEEDING

Significant bleeding is a rare complication of diagnostic upper endoscopy. Bleeding may be more likely in individuals with thrombocytopenia and/or coagulopathy. However, diagnostic upper endoscopy appears to be safe in patients with platelet counts as low as 20,000.1 Biopsies should be performed with caution below this level and platelet transfusions should be considered. Mallory-Weiss tears occur in <0.1% of diagnostic endoscopies and are usually not associated with significant bleeding [64].

STRICTURE AND FISTULA

These are known to occur following diagnostic and therapeutic modalities. Esophageal stricture formation can occur weeks to months after EVS sessions in 2% to 20% of patients. This can be diagnosed by upper gastrointestinal series and/or endoscopy [64].

ASPIRATION PNEUMONIA

Up to 5% of patients may experience aspiration pneumonia after EVS[64].

Complications are known to occur in the following settings :

a) Esophageal dilatation of benign / malignant strctures and achalasia

b) PEG placement

c) During endoscopic foreign body removal.

d) During the treatment of esophageal malignancies by the mode of photodynamic therapy and also following endoprostheses placement (stent migration , hemorrhage and food impaction)

e) During endoscopic hemostatic procedures like endoscopic variceal sclerotherapy and endoscopic band ligation [64].

ADVANCES IN ENDOSCOPY- ERCP

INTRODUCTION

ERCP was first reported in 1968 [65] and was soon accepted as a safe, direct technique for evaluating biliary and pancreatic disease. With the introduction of endoscopic sphincterotomy in 1974 [66], therapeutic pancreaticobiliary endoscopy subsequently was developed. ERCP is now widely available.

ERCP IN BILIARY TRACT DISEASE

ERCP is particularly useful in the management of the jaundiced patient with biliary obstruction because of choledocholithiasis and strictures.

Successful endoscopic cholangiography with relief of obstruction should be technically achievable in more than 90% of patients. Cholangioscopy at ERCP is used infrequently but may be helpful in the management of bile-duct stones and in assessing suspected malignancies [67].

Choledocholithiasis

The most common source of biliary obstruction is choledocholithiasis. Such patients may present with biliary colic, obstructive jaundice, cholangitis, or pancreatitis. The sensitivity and the specificity of ERCP for detecting common duct stones is over 95%;small stones occasionally are missed [68].

Therapy for choledocholithiasis

Endoscopic sphincterotomy and stone extraction is successful in more than 90% of cases, with an overall complication rate of approximately 5% and a mortality rate of less than 1% in expert hands [69].

An alternative to biliary sphincterotomy is balloon dilation of the biliary sphincter (balloon sphincteroplasty). This may be an alternative to biliary sphincterotomy in selected patients with common bile duct stones, e.g., underlying coagulopathy, albeit with a higher risk of post-ERCP pancreatitis [70, 71].

ERCP IN MALIGNANT AND BENIGN BILIARY STRICTURES

ERCP is useful in the assessment and the treatment of malignant biliary obstruction.Biopsies, brushings, and FNA may yield a definitive tissue diagnosis, but the combined sensitivity is no higher than 62% [72,73].

ERCP is indicated for the evaluation and the treatment of benign bile-duct strictures, congenital bile-duct abnormalities, and postoperative complications. This applies to patients with biliary obstruction after liver transplantation [74, 75].

Stricture dilation

Benign biliary strictures may be dilated with hydrostatic balloons or a graduated catheter passed over a guidewire.

Indications for endoscopic dilation of benign strictures include postoperative strictures, dominant strictures in sclerosing cholangitis, chronic pancreatitis, and stomal narrowing after choledochoenterostomy [76].

Stent placement may be used to maintain patency after initial dilation when using single or multiple endoscopic prostheses [77].

Stents

Endoscopically placed bile-duct stents have a role in the treatment of both malignant and benign biliary strictures, as well as in postoperative bile-duct injuries or leaks [78].

ROLE OF ERCP IN PANCREATIC DISEASE [79]

A. ERCP plays an important role in patients with recurrent acute pancreatitis and can identify and, in some cases, treat underlying causes.

B. ERCP is effective in treating symptomatic strictures in chronic pancreatitis.

C. ERCP is effective for the palliation of malignant biliary obstruction, for which self-expanding metallic stents have longer patency than plastic stents.

D. ERCP can be used to diagnose and to treat symptomatic pancreatic-duct stones.

E. Pancreatic-duct disruptions or leaks can be effectively treated via the placement of bridging or transpapillary pancreatic stents.

F. ERCP is a highly effective tool to drain symptomatic pancreatic pseudocysts and, in selected patients, more complicated benign pancreatic-fluid collections arising in patients with a history of pancreatitis.

G. Intraductal US and pancreatoscopy are useful adjunctive techniques for the diagnosis of pancreatic malignancies [79].

ENDOSCOPIC ULTRASONOGRAPHY

Advances in ultrasound technology led to transducer miniaturization, allowing for intra-coronary sonography in 1989 [80]. In the same year, initial experience with a mechanical linear ultrasound probe for evaluation of gastrointestinal use in the canine model was reported [81]. High frequency ultrasound probe sonography (HFUPS) has aroused interest because it can be performed through the biopsy channel of an endoscope providing ultrasound imaging of visible lesions without the need for endoscope exchange.

Technical Considerations

Ultrasound probes for endoscopic use are available as 2, 2.4 and 2.6 mm in with frequencies of 12, 15 and 20MHz and in lengths up to 220 cms. These high frequencies allow for detailed resolution of the gastrointestinal wall at the expense of depth of penetration. Reported mean imaging depths for the 12 MHz and 20 MHz probes are 29 mm and 18 mm respectively [82, 83].

Probes consist of a mechanical rotating scanner with a protective cover, filled inside with oil that serves as an acoustica interface providing a 360. radial image in a plane perpendicular to the probe axis. Mechanical linear images can also be obtained with one of the available systems. Scanning is

performed with the water immersion method or a balloon sheath placed over the probe.

In contrast to the five-layered gastrointestinal wall structure obtained with conventional EUS, HFUPS can delineate the 7 to 9 layered structure. The muscularis mucosae, not normally seen, is visualized as two layers in up to 70% of cases and the Muscularis propria as a three layered structure, circular layer, interface and longitudinal layer. This could have significant clinical relevance for endoscopic mucosal resections (EMR) or in the evaluation of motility disorders [84 - 87].

Applications

HFUPS has been used in the staging of esophageal, gastric, ampullary, pancreatobiliary, and colonic neoplasms. Reported accuracy in staging superficial esophageal carcinoma, early gastric cancer and flat colorectal tumors were 85%, 67 to 72.3% and 76 to 88% respectively. Local and peri-tumoral lymph nodes can be detected by HFUPS, but similar to conventional EUS cannot differentiate between benign of malignant nodes. Unlike conventional EUS, regional or deeper nodes cannot be visualized by HFUPS making it inadequate for TNM staging .Another application for HFUPS is in selecting the subgroup of patients with superficial neoplasms who are candidates for Endoscopic Mucosal Resection (EMR) [84-87].

Staging of ampullary and pancreaticobiliary malignancies has been reported by placing the ultrasound probes into the common bile duct and pancreatic ducts also referred as intraductal ultrasonography IDUS) [88, 89].

DYSPEPSIA

DEFINITION

"Dyspepsia is defined as a constellation of symptoms that include upper abdominal pain or discomfort, which is intermittent or constant and may be associated with additional symptoms of nausea and vomiting [90]."

Although these symptoms may be associated with a wide range of specific clinical diagnoses (peptic ulcer disease [PUD], gastric cancer, and gastroesophageal reflux [GERD], among others), often no organic cause can be found (functional dyspepsia) [91].

Organic *versus* idiopathic dyspepsia.

From an etiological viewpoint, patients with dyspeptic symptoms can be subdivided into 2 main categories [92]:

1. Those with an **identified** organic or metabolic cause for the symptoms where, if the disease improves or is eliminated, symptoms also improve or resolve (eg, peptic ulcer disease, GERD with or without esophagitis, malignancy, pancreaticobiliary disease, or medication use).

2. Those with **no identifiable** explanation for the symptoms. In some of these patients, an identifiable pathophysiological or microbiologic abnormality of uncertain clinical relevance (eg, *Helicobacter pylori* gastritis) may be present, which is not thought to explain the symptoms. Others have abnormal motor or sensory dysfunction (eg, altered gastric emptying, fundic dysaccommodation, or gastroduodenal hypersensitivity) of uncertain significance. This broad group of patients with idiopathic dyspepsia has previously been referred to as nonulcer dyspepsia, essential dyspepsia, idiopathic dyspepsia, or Functional Dyspepsia (FD). FD is currently the most recognized term in the literature.

DYSPEPTIC SYMPTOMS AND THEIR DEFINITIONS [92]

Epigastric pain

Epigastric refers to the region between the umbilicus and lower end of the sternum, and marked by the midclavicular lines. Pain refers to a subjective, unpleasant sensation; some patients may feel that tissue damage is occurring. Other symptoms may be extremely bothersome without being interpreted by the patient as pain.

Epigastric burning

Epigastric refers to the region between the umbilicus and lower end of the sternum, and marked by the midclavicular lines. Burning refers to an unpleasant subjective sensation of heat.

Postprandial fullness

An unpleasant sensation like the prolonged persistence of food in the stomach

Early satiation

A feeling that the stomach is overfilled soon after starting to eat, out of proportion to the size of the meal being eaten, so that the meal cannot be finished. Previously, the term “early satiety” was used, but satiation is the correct term for the disappearance of the sensation of appetite during food ingestion [92].

CAUSES OF ORGANIC DYSPEPSIA [93]

A. Luminal GI tract

a) Food intolerance

b) Peptic ulcer disease

c) Gastroesophageal reflux

d) Gastric or esophageal neoplasms

e) Gastroparesis (diabetes, postvagotomy, scleroderma, chronic intestinal pseudo-obstruction)

f) In.ltrative gastric disorders (Menetrier's syndrome, Crohn's disease, eosinophilic gastroenteritis, sarcoidosis, amyloidosis)

g) Malabsorptive disorders (celiac sprue, lactose intolerance)

h) Gastric infections (CMV, fungal, TB, syphilis)

i) Parasites (*Giardia lamblia, Strongyloides stercoralis)*

j) Chronic gastric volvulus

k) Chronic intestinal ischemia

l) Irritable bowel syndrome

B. INTOLERANCE TO MEDICATIONS [93]:

- Ethanol, Aspirin/NSAIDs
- Antibiotics (macrolides, sulfonamides, metronidazole)
- Theophylline ,Digitalis, Glucocorticoid

- Iron, potassium chloride, Niacin, gemfibrozil
- Narcotics, Colchicine, Quinidine
- Estrogens, Levodopa
- Nitrates, Loop diurectics, ACE inhibitors

C. PANCREATICOBILIARY DISORDERS [93]:

- Chronic pancreatitis
- Pancreatic neoplasms
- **Biliary colic:** cholelithiasis, choledocholithiasis, sphincter of Oddi dysfunction

D. SYSTEMIC DISORDERS [93]:

- Diabetes mellitus
- Thyroid disease
- Hyperparathyroidism
- Adrenal insuf.ciency
- Collagenvascular disorders
- Renal insuf.ciency
- Cardiac ischemia, congestive heart failure
- Intra-abdominal malignancy
- Pregnancy

FUNCTIONAL DYSPEPSIA [92]

A large group of patients with functional gastrointestinal disorders have chronic symptoms that can be attributed to the gastroduodenal region . Based on the consensus opinion of an international panel of clinical investigators who reviewed the available evidence, functional gastroduodenal disorders were classified as follows:

CATEGORY B. FUNCTIONAL GASTRODUODENAL DISORDERS

B1. Functional dyspepsia

B1a. Postprandial distress syndrome

B1b. Epigastric pain syndrome

B2. Belching disorders

B2a. Aerophagia

B2b. Unspecified excessive belching4

B3. Nausea and vomiting disorders

B3a. Chronic idiopathic nausea

B3b. Functional vomiting

B3c. Cyclic vomiting syndrome

B4. Rumination syndrome in adults

B1. DIAGNOSTIC CRITERIA* FOR FUNCTIONAL DYSPEPSIA [92]

Must include:

1. ***One or more* of:**

a. Bothersome postprandial fullness

b. Early satiation

c. Epigastric pain

d. Epigastric burning

AND

2. No evidence of structural disease (including at upper endoscopy) that is likely to explain the symptoms.

(*Criteria fulfilled for the last 3 months with symptom onset at least 6 months before diagnosis)

B1a. DIAGNOSTIC CRITERIA* FOR POSTPRANDIAL DISTRESS SYNDROME [92]

Must include *one* ***or*** *both* **of the following:**

1. Bothersome postprandial fullness, occurring after ordinary sized meals, at least several times per week.

2. Early satiation that prevents finishing a regular meal, at least several times per week.

(*Criteria fulfilled for the last 3 months with symptom onset at least 6 months before diagnosis)

Supportive criteria

1. Upper abdominal bloating or postprandial nausea or excessive belching can be present.

2. EPS may coexist.

B1b. DIAGNOSTIC CRITERIA* FOR EPIGASTRIC PAIN SYNDROME [92]

Must include *all* of the following:

1. Pain or burning localized to the epigastrium of at least moderate severity at least once per week.

2. The pain is intermittent.

3. Not generalized or localized to other abdominal or chest regions.

4. Not relieved by defecation or passage of flatus.

5. Not fulfilling criteria for gallbladder and sphincter of Oddi disorders.

(*Criteria fulfilled for the last 3 months with symptom onset at least 6 months before diagnosis.)

SUPPORTIVE CRITERIA:

1. The pain may be of a burning quality but without a retrosternal component.

2. The pain is commonly induced or relieved by ingestion of a meal but may occur while fasting.

3. Postprandial distress syndrome may coexist.

PATHOPHYSIOLOGY

The pathophysiologic characteristics of symptoms of functional dyspepsia are poorly understood. Dyspepsia is considered to be part of a continuum of functional GI disorders that involve the entire gut.

A. ABNORMALITIES IN GASTRODUODENAL MOTILITY

1. Delayed Gastric Emptying

Delayed gastric emptying measures the integrated efficiency of gastric neuromuscular work in response to a meal [94].

2. Impaired Gastric Accommodation

Ultrasonography, scintigraphy, and barostats have shown that in contrast to normal subjects, whose food is initially accommodated in the fundus and body with gradual redistribution to the antrum, over 40% of patients with functional dyspepsia have impaired accommodation of the proximal stomach, which may lead to early distribution of food to the distal stomach with dilatation of the antrum [95, 96].

3. Myoelectrical Abnormalities

Noninvasive cutaneous electrogastrography (EGG) can measure fasting and postprandial gastric electrical activity. The basal electrical rhythm (BER) is generated by a pacemaker located in the proximal body and propagated longitudinally and circumferentially at a normal rate of 3 cycles per minute. Gastric dysrhythmias are identified by EGG in 40% of patients with functional dyspepsia but also in 20% of normal controls [97, 98].

B. VISCERAL HYPERSENSITIVITY

* Afferent stimulation of gut mechanoreceptors reaches conscious perception through a three-neuron chain.

* Descending fibers from brainstem centers modulate the sensitivity of the dorsal horn neurons and control the perception of visceral sensation [99, 100].

* Most stimuli arising from the GI tract (accommodation, gastric emptying, distention, contractions) are not consciously perceived; however, a lowering of the perception threshold may occur in patients with functional dyspepsia, generating heightened sensitivity to normal physiologic events or minor noxious stimuli.61 Hypersensitivity to distention of the stomach can be demonstrated in more than 50% of patients with functional dyspepsia, both those who seek medical attention and non-consultors [101].

C. AUTONOMIC NEUROPATHY

The vagus nerve regulates gastric accommodation and emptying and exerts a visceral antinocioceptive effect [101].

It is hypothesized that acute and chronic life stresses and psychological factors may lead to decreased vagal tone, which results in the pathophysiologic abnormalities that give rise to dyspeptic symptoms [102].

D. HELICOBACTER PYLORI

The prevalence of *H. pylori* infection in patients with functional dyspepsia is similar to that in the general population.

The strongest evidence against the role of *H. pylori* in functional dyspepsia is that controlled therapeutic trials of *H.pylori* eradication demonstrate no significant long-term improvement in symptoms [103].

E. PSYCHOSOCIAL FACTORS

The degree to which psychosocial factors are contributing and remediable should be assessed in every functional dyspepsia patient [104].

For many patients with functional dyspepsia, abdominal symptoms are part of a constellation of somatic and psychological complaints [105].

ROLE OF H.PYLORI IN DYSPEPSIA [106]

Treatment of non-investigated dyspepsia may be different if the incidence of *H. pylori* is as low as occurs in developed countries. The increasing awareness of *H. pylori* as a pathogen in developing countries has stimulated interest in a test-and-treat approach in these areas. A test-and-treat approach was recommended in adult patients below 45 years of age – the age cut-off may vary locally – presenting in primary care with persistent dyspepsia having excluded those with predominantly gastrooesophageal reflux disease (GORD), non-steroidal anti-inflammatory drugs (NSAIDs) consumption and those with alarm symptoms. This recommendation has been vindicated in more recent publications. The definition of low prevalence is a population with an infection rate of less than 20% [106].

RECOMMENDATIONS

- *H. pylori* test and treat is an appropriate option for patients with non-investigated dyspepsia.
- *H. pylori* eradication is an appropriate option for patients infected with *H. pylori* and investigated non-ulcer dyspepsia.

- *H. pylori* test and treat is the strategy of choice in all (adult) patients with functional dyspepsia in high-prevalence populations.

- The effectiveness of *H. pylori* test and treat is low in populations with a low *H. pylori* prevalence. In this situation, the test-and-treat strategy or empirical acid suppression are appropriate options [106].

TREATMENT

TREATMENT OF FUNCTIONAL DYSPEPSIA: [93]

Most functional dyspepsia patients have intermittent, mild symptoms that respond to reassurance and life-style modifications. Refractory symptoms, however, may be difficult to manage. Lack of improvement may lead to concern that an organic cause has been overlooked and to repeated testing.

Multicenter, Randomized Trials in Funcational Dyspepsia Comparing Resolution of Dyspepsia in Patients Treated with Proton Pump Inhibitors, Ranitidine, or Placebo.

STUDY (REFERENCE)	TREATMENT (DOSE)	%TREATMENT SUCCESS
"Bond" Study	Omeprazole (20 mg/day) Omeprazole (10 mg/day Placebo	43% 43% 26%
"Opera" Study	Omeprazole (20mg/day) Omeprazole (10 mg/day) Placebo	34% 30% 31%
Combined studies	Omeprazole (20 mg/day) Omeprazole (10 mg/day) Placebo	38% 36% 28%

ANTISECRETORY AGENTS

Antisecretory therapies —both H2-receptor antagonists and proton pump inhibitors—are useful in a subset of patients with functional dyspepsia, primarily those with heartburn or significant epigastric pain, and an empirical trial of such agents is reasonable. It is unlikely that they afford any significant benefit to patients with other dyspeptic symptoms.

In the subsets who benefit, it has not been established that proton pump inhibitors are superior to less expensive H2-receptor antagonists [93].

PROMOTILITY AGENTS:

Promotility agents decrease gastroesophageal reflux, improve gastric emptying, and facilitate accommodation and might thereby be predicted to benefit some patients with functional dyspepsia.

A. Domperidone, a peripherally acting dopaminergic antagonist does not cross theCNS blood-brain barrier, may be used, and it has shown considerable benefit.

The high incidence of adverse CNS effects and extrapyramidal effects associated with metoclopramide makes it unsuitable for long-term use.

Cisapride has been markedly restricted in its use by the United States Food and Drug Administration because of a low but significant risk of QT prolongation and cardiac tachyarrhythmias and should no longer be prescribed for functional dyspepsia [93].

TREATMENT OF H.PYLORI

The European Consensus suggested that screening for *H pylori* followed by eradication therapy should be given to all dyspeptic patients younger than 45 years.

BISMUTH-BASED TRIPLE THERAPY

Bismuth compounds have been used for decades to treat dyspepsia and peptic ulceration—even before the anti–*H pylori* action of the compounds was known. The classic triple therapy of bismuth (colloidal bismuth subcitrate or bismuth subsalicylate), metronidazole, and either amoxycillin or tetracycline is the most common regimen. Tetracycline-containing triple therapy achieves a greater cure rate than the amoxicillin alternative. With a 1-week course of triple therapy, both duodenal and gastric ulcers heal—even without acid suppression by H2-receptor antagonists or proton pump inhibitors [107].

DUAL THERAPY

Dual therapy refers to the combination of PPIs or ranitidine bismuth citrate (RBC) and one antibiotic, usually amoxycillin or clarithromycin. Inhibition of acid secretion with a PPI or H2-receptor antagonist increases the intragastric acid level to pH5 or more and acts synergistically with amoxycillin and clarithromycin.

These regimens are better tolerated and simpler to follow than bismuth-based triple therapy. The first dual therapy combining omeprazole with amoxycillin had unpredictable efficacy ranging from 20% to 90% and thus credibility with most gastroenterologists [107].

TRIPLE THERAPY

To date, the most popular treatment regimen for the cure of *H pylori* infection consists of an acid-suppressant (PPI or RBC) and two antimicrobial

agents PPI-triple therapy, no difference in the cure rate of *H pylori* infection and duodenal ulcer was found. One-week RBC-based triple therapy is now increasingly [107].

The Metronidazole, Amoxycillin, Clarithromycin, *Helicobacter* **(MACH)-1** study tested omeprazole in combination with various antimicrobials (amoxycillin, tetracycline, and metronidazole) and confirmed the efficacy of this 1-week regimen. The best results were obtained from the therapies of omeprazole, clarithromycin, and amoxycillin or metronidazole. Their side effects are much milder than the original bismuth based triple therapy and patient compliance is expected to improve. The role of omeprazole in these non– bismuth-based triple therapies has been substantiated by the **MACH-2** study; the role appears to be a class effect of PPI.

The choice of antibiotics decides the efficacy of PPI-based triple therapy [107].

QUADRUPLE THERAPY

Quadruple therapy combines an acid-suppressive drug, usually a PPI, with three antimicrobial agents. Typical quadruple therapy includes omeprazole, tetracycline, metronidazole, and a bismuth salt. Newer quadruple therapy may comprise another PPI, amoxycillin, clarithromycin, and metronidazole. Studies have been done to evaluate the possible role of quadruple therapy in shortening the duration of treatment or improving the efficacy of eradication [107].

AIM OF THE STUDY

1. To evaluate patients with significant upper gastrointestinal symptoms by doing an UGI-Scopy and analysis the various causes.

2. To correlate symptoms with Endoscopic findings to determine the significance of a symptom as an indicator of diseasaes.

3. To determine the importance of smoking, alcohol and NSAID's as etiological factors of upper GI disease as shown by UGI-scopy.

MATERIALS AND METHODOLOGY

SELECTION CRITERIA OF SUBJECTS

Patients attending both medical / Medical gastroenterology OPD with UGI symptoms during the period from Sep. 2004 to Sep. 2006 as a prospective study at Kilpauk Medical College and Hospital,Chennai.

INCLUSION CRITERIA

1. All patients presenting with UGI symptoms of pain abdomen (epigastric), heart burn, early satiety, bloating, vomiting, nausea, anorexia, loss of appetite, dysphagia and GI bleeding (for a period of more than 20 days).

2. All patients of either sex above 15 years of age.

3. Only those patients who consented for the procedure and underwent the same.

EXCLUSION CRITERIA

1. Age less than 15 years.

2. Patients with cirrhosis, portal hypertension, corrosive poisoning or those undergoing renal transplant program, pre-surgical evaluation of umbilical hernia, gall stone disease or any other abdominal symptoms apart from UGI-symptoms.

3. Patients undergoing follow up or a therapeutic endoscopy.

METHODOLOGY

- All patients were informed about the nature of the procedure and consent was obtained for the same.

- Patients who underwent UGI scopy on an elective list, were advised to come on an overnight fast or atleast a fast of 6-8 hrs minimum. The same will be applicable to inpatients also.

- Dentures and spectacles were removed.

- Patients were not sedated.

- Cardiac assessment was obtained for patients where it was necessary.

- Clinical monitoring of BP, Pulse, Respiratory rate, Oxygen Saturation during and after procedure in some cases as deemed necessary was done.

- Pharyangeal anaesthetic solution was given for gargling just before the procedure.

ENDOSCOPY

U.G.I.Scopy was done using a PENTAX for viewing fibre optic endoscopic. Only endoscopic findings were considered. Thus the diagnosis of oesophageal or gastric cancer was based on the presence of endoscopic features traditionally suggestive of malignancy. Oesophagitis was diagnosed according to criteria of Savary and Miller. In the absence of endoscopy stigmata of malignancy, gastric ulcer was considered as benign.

The upper GI-symptoms with which the patients presented including upper abdominal pain, nausea, vomiting, heart burn, early satiety, bloating, dysphagia, weight loss, GI-bleed and anorexia were recorded.

Details of tobacco use (smoking) alcohol, NSAIDS usage were recorded (including duration and frequency).

UGI endoscopy was performed and findings were recorded as follows:

1. Normal study
2. Oesophagitis/Oesophageal ulcer
3. Carcinoma Oesophagus.
4. Gastritis
5. Gastric Ulcer
6. Carcinoma Stomach
7. Duodenal ulcer / Duodenitis
8. Duodenal Stricture / GOO

STATISTICAL ANALYSIS

Univariate analysis of each of the symptoms was correlated with the endoscopy outcome. Statistical analysis was done using SPSS, Software and findings confirmed with the help of statistician.

OBSERVATIONS

Data collected from 281 patients selected for study based on the inclusion criteria were analysed . Using the proforma the patients age, sex and personal habits were documented . All the presenting complaints of the patients, pertaining to the upper gastrointestinal system and their duration were also noted down.

Patients selected for endoscopy were those with symptoms for atleast 20 days, irrespective of previous history of treatment for these symptoms (except in the case of malena / haemetemisis where patient under went UGI-endoscopy at the earliest.

PATIENT DEMOGRAPHY

The study population consisted of 281 patients with a mean age of 44 years . The youngest patient was 15 years old and the oldest 85 years . There were 76 females and 87 males below the age of 45 years who under went UGI scopy. But on the whole out of 281 patients, 49.8% were males and 50.2% were females.

AGE DISTRIBUTION OF PATIENTS

AGE IN YEARS	MALE	FEMALE	TOTAL	%
15 to 30	41	32	73	25.97
30 to 45	46	44	90	32
45 to 60	25	39	64	22.8
>60	28	26	54	19.21
	140	141	281	

TABLE - 1

Among the 281 patients 33.8% were smokers and 14.6% were alcoholics,whereas 13.52% consumed both. Approximately 28% were consuming NSAID,s regularly.

SYMPTOMS VS DISEASE DISTRIBUTION

S. NO	SYMPTOMS	% OF PATIENTS	NO. OF PATIENTS	NORMAL STUDY	ESOPHAGITIS	CA. ESOPHAGUS	GASTRITIS	GASTRIC ULCER	CA. STOMACH	DUODENAL ULCER	DUODENITIS	DU SCARRING / GOO
1	UPPER ABDOMEN PAIN	70.5	198	58	60	N	63	5	1	18	30	6
2	HEART BURN	51.6	145	42	48	N	51	6	N	8	21	2
3	NAUSEA	43.4	122	36	35	N	42	4	2	8	16	5
4	VOMITING	34.5	97	24	31	1	32	1	2	7	14	5
5	LOSS OF APPETITE	32.4	91	26	30	3	26	1	3	8	10	4
6	BLOATING	18.9	53	20	13	N	14	2	2	4	8	4
7	BELCHING	16.4	46	14	15	N	15	2	N	4	7	3
8	EARLY SATIETY	14.9	42	16	10	N	12	1	3	3	4	3
9	DYSPHAGIA	7.8	22	9	6	3	4	N	1	1	N	N
10	ANAEMIA	7.5	21	4	4	1	7	1	1	5	3	1
11	UGI – BLEED	6	17	2	1	N	7	2	N	7	4	N
12	LOSS 0F WEIGHT	5	14	3	N	3	2	N	3	2	N	2

TABLE - 2

ANALYSIS OF ENDOSCOPIC FINDINGS

- Out of the 281 patients who presented with atleast one of the UGI symptoms 33.8% did not have any upper GI disease on endoscopy.
- The most common finding on UGI scopy was gastritis ,30%, followed by reflux esophagitis at 26%.
- Duodenal ulcer was 4 times more than gastric ulcer (25 vs 6). Approximately 12% also had duodenitis.
- Out of the 84 patients who had gastritis, 75% presented with abdomen pain, 61% had heart burn and 50% of them had nausea.
- Abdomen pain, heart burn, nausea and vomiting were the most common presentations in that order in patients diagnosed with esophagitis.
- Among the 31 patients who had peptic ulcer disease (25-duodenal ulcer, 6- gastric ulcer), apart from abdomen pain and heart burn being the most common presentations, one third had lossof appetite,another one third presented with UGI bleed(malena / haematemesis), and one fourth of them presented with anaemia.
- Patients who had duodenal disease most commonly presented with upper abdomen pain.
- Out of the six patients diagnosed with malignancy,3 had carcinoma esophagus and 3 carcinoma stomach.
- Seven patients were diagnosed to have duodenal stricture , among which 4 patients developed gastric outlet obstruction, all of them males, who were regular smokers.

ANALYSIS OF UGI SYMPTOMS AT PRESENTATION

- Upper abdomen pain was the most common complaint at presentation at 70%.
- The next common presentation was heart burn (51.6%).
- Nausea and vomiting were observed at 43.4% and 34.5% respectively.
- Approximately 32% of the patients presented with loss of appetite.
- One out of every five patients presented with bloating, belching, and or early satiety.

PREDICTORS OF ENDOSCOPIC FINDINGS WITH RESPECT TO DYSPEPTIC SYMPTOMS

TABLE - 3

S.No	SYMPTOMS	UGI FINDINGS PRESENT		Odds Ratio	95% CI	p value (Pearson Chi-square)
		Symptom Present (%)	Symptom Absent (%)			
1	Upper abdomen pain	70.7	55.4	1.94	1.14 - 3.29	0.013(Sig)
2	Heart burn	71	61	1.56	0.95 - 2.57	0.076(ns)
3	Nausea	70.5	62.9	1.41	0.85 - 2.33	0.182(ns)
4	Vomiting	75.3	61.4	1.91	1.10 - 3.29	0.020(Sig)
5	Loss of appetite	71.4	63.7	1.42	0.83 - 2.44	0.199(ns)
6	Bloating	62.3	67.1	0.80	o.44 - 1.49	0.502(ns)
7	Belching	69.6	65.5	1.20	0.61 - 2.36	0.597(ns)
8	Early satiety	61.9	66.9	0.80	0.41 - 1.57	0.524(ns)
9	Dysphagia	59.1	66.8	0.718	0.30 - 1.70	0.463(ns)
10	Anaemia	81	65	2.28	0.78 - 6.67	0.137(ns)
11	UGI bleed	88.2	64.8	4.079	1.01 – 16.3	0.047(Sig)
12	Loss of weight	78.6	65.5	1.93	0.562 – 6.56	0.315(ns)

Among 198 patients who presented with **upper abdomen pain** 71% had UGI disease and of those without abdomen pain 55.4% had positive UGI findings. This difference was statistically significant (p value of 0.013).

TABLE - 4

UPPER ABDOMEN PAIN	UGI DISEASE PRESENT	UGI DISEASE ABSENT	TOTAL	%
Present	**140**	**58**	**198**	**70.7**
Absent	**46**	**37**	**83**	**55.4**
	186	**95**	**281**	

p value = 0.013

Vomiting was found to be a statistically significant predictor of upper GI disease (p value of 0.020). Out of the 97 patients who presented with vomiting, one-third had esophagitis and one-third had gastritis, whereas 25% were not found to have any UGI disease on endoscopy.

TABLE - 5

VOMITING	UGI DISEASE PRESENT	UGI DISEASE ABSENT	TOTAL	%
Present	**73**	**24**	**97**	**75.3**
Absent	**113**	**71**	**184**	**61.4**
	186	**95**	**281**	

p value= 0.020

- Among 74 patients with esophagitis, 57% presented with heart burn.

- Seventeen patients presented with UGI bleed in the form of either malena or haematemesis or both, out of which 7 had doudenal ulcer, 2 patients had gastric ulcer and another seven patients had gastritis. Upper GI bleed was found to be a significant predictor of disease on endoscopy.

- Upper GI endoscopy was indicated and done in 21 patients with anaemia, among whom 16 had UGI disease. For 9 of them the cause was in the stomach (7-gastritis,1-gastric ulcer, 1-carcinoma stomach), and the rest of them had duodenal disease (7-duodenal ulcer, 4-duodenitis).

- Both loss of weight and loss of appetite were significant symptoms for predicting upper GI malignancy (Ca.stomach / Ca. esophagus).

ANALYSIS OF THE INFLUENCE OF SMOKING ALCOHOL AND NSAID'S ON UPPER GI DISEASE

S.NO	HABITS	% OF PATIENTS	NO. OF PATIENTS	NORMAL STUDY	ESOPHAGITIS	CA. ESOPHAGUS	GASTRITIS	GASTRIC ULCER	CA. STOMACH	DUODENAL ULCER	DUODENITIS	DU SCARRING / GOO
1	SMOKING	33.8	95	17	33	N	33	4	N	12	18	6
2	ALCOHOL	14.6	41	9	14	N	16	4	N	N	7	1
3	NSAID's	23.1	65	14	20	N	24	2	N	8	11	N

TABLE -6

PREDICTORS OF ENDOSCOPIC FINDINGS WITH RESPECT TO HABITS

S.No	HABITS	UGI Findings Present		Odds Ratio	95% CI	p value (Pearson Chi-square)
		Habits Present (%)	Habits Absent (%)			
1	SMOKING	82.1	58.1	3.31	1.82–6.00	<0.00003 (Sig)
2	ALCOHOL	78	64.2	1.98	0.91–4.28	0.082(ns)
3	NSAID'S	78.5	62.5	2.18	1.14–4.16	0.017(Sig)

TABLE –7

33.8% of patients were smokers . 82% of smokers had disease, whereas only 58% of non-smokers, had UGI disease. The difference was statistically significant with a p value of < 0.0001.

TABLE - 8

SMOKING	UGI DISEASE PRESENT	UGI DISEASE ABSENT	TOTAL	%
Present	78	17	95	82.1
Absent	108	78	186	58.1
	186	95	281	

p value <0.0001

NSAID's usage was a significant factor in the causation of upper GI disease, with disease rates of 78.5% among those who regularly used them and the p value being statistically significant at 0.017.

TABLE -9

NSAID's USAGE	UGI DISEASE PRESENT	UGI DISEASE ABSENT	TOTAL	%
Present	**51**	**14**	**65**	**78.5**
Absent	**135**	**81**	**216**	**62.5**
	186	**95**	**281**	

p value = 0.017

DISCUSSION

The term dyspepsia is used variably by health professionals to refer to a heterogeneous group of upper abdominal symptoms that may arise from numerous causes [108] .

Regardless of numerous studies and trials dyspepsia still remains a controversial issue.

In recent studies, the focus has been placed on the predominant symptom as a possible indicant of the underlying disorder, instead of classifying into symptom complexes and unspecified sub groups [109, 110].

The advent of endoscopy has caused a sea of change in the definitions and management of dyspepsia , following which a new entity called functional dyspepsia has been defined, thanks to endoscopy.

This study included 281 patients with a mean age 44 years, 58% were below the age of 45 years and 42% above 45 years .

Abdomen pain was the most common presenting symptom seen in 198 patients constituting around 70.5% , and it turned out to be a significant predictor of upper gastrointestinal disease. This is comparable to a study by Kolk H where 73% of the patients had upper abdominal pain as the most common presenting symptom [111].

Globally the prevalance of functional dyspepsia has been noted to vary between 11% to 29.2%. In this study around 33% of patients referred for endoscopy did not have detectable upper GI disease being at par [112].

33.8% of the patients were smokers in this study and it was found that it was positively associated with with upper GI disease. A study by Moshkowitz M et al, has similarly comparable findings and concludes that the

incidence of significant upper GI disease is more prevalant in smokers with a p value of less than 0.05 [113].

There were 23.1% of patients in this study who had a history of regular NSAID's usage and this was significantly attributable with upper GI disease. There were many studies confirming the same world over in different measures one among them was a study conducted by Devi DP et al in a south Indian hospital which implicates the NSAID's to the same measure (p value <.005) [114].

Out of 97 patients who presented with vomiting 75% had positive endoscopic findings one third of whom had reflux esophagitis.

Also out of the 145 patients who had presented with heart burns one third had reflux esophagitis and another one third had gastritis.

UGI bleeding is the only alarm symptom that definitely had positive disease on endoscopy.

Loss of weight and loss of appetite were significant predictors of malignancy alone, as conformed by Adang et al [115].

SUMMARY

- 281 patients presented with dyspeptic symptoms.
- Age of the patients varied from 15 to 85 with a mean age of 44.
- 163 patients (58%)were below the age of 45 years and 118 patients (42%) were above the age of 45 years.
- Among the dyspeptic symptoms upper abdomen pain, vomiting and UGI-bleed(malena/ haemetemesis)were significant predictors of upper GI disease.
- One third of patients with heart burns had relux esophagitis.
- Roughly one third of the patients who presented with dyspeptic symptoms did not have any detectable ugi disease on endoscopy.
- Loss of weight and loss of appetite were significantly associated with upper GI malignancy.
- Smoking habits and NSAID's usage were definitely found to be causative factors of upper GI disease.

CONCLUSION

Acid peptic disease still remains a common condition in the southern part of the country. The most common presenting symptoms are upper abdomen pain, heart burn, nausea and vomitting. Gastrititis, Reflux disease, duodenitis and peptic ulcer are the most common conditions we see.

Although malignancy is one of the possibilities it remains to have a low incidence compared to ulcer disease.

The evaluation of symptoms to the pathology in the upper GI tract shows a significant correlation of abdomen pain and heart burn to oesophagitis and abdomen pain, heart burn, nausea and vomiting to gastritis.

The need for an endoscopy of the upper GI tract can never be over emphasized keeping in mind the need for proper therapy. It also serves to alleviate the fear of a serious disorder and calm the patient. However endoscopy requires costly equipment, technical capability and a willing patient. Some patients could not be included as they were not willing for endoscopy. In this situation the attending physician is forced to treat symptomatically.

On the basis of this study the combination of upper abdomen pain and heartburn was seen in a large percentage of patients having reflux oesophagitis and gastritis. Therefore these patients can for practical purposes be treated for reflux disease even without a scopy (provided we are sure we will not be missing a malignancy). This would not only be applied to reluctant patients but would reduce hospital costs and the medical personnel would have more time available. The period of medical treatment can be given for twenty one days and patients reviewed in the out patient department.

History is of paramount importance in the diagnosis and treatment of upper GI disease. It will help us to exclude certain conditions and zero-in an the diagnosis, on a clinical basis. However endoscopy remains the most valuable tool for further evaluation.

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APPENDIX

PROFORMA

Name : Occupation :

Age & Sex: Education :

I.P.No.

UGI Endoscopy No

Date of Endoscopy

HISTORY OF : 1 / 2 (Preceding 1 Yr)

Tobacoo (Smoking, Chewing, etc.)

Alcohol : 1 / 2 (Preceding 1 week)

NSAID Use : 1 / 2 (Preceding 1 week)

Others

DIABETIC : 1 / 2

HYPERTENSIVE : 1 / 2

SYMPTOMS AT PRESENTATION

a) Upper Abdomen pain / Discomfort : 1 / 2

b) Heart Burn : 1 / 2

c) Early Salary : 1 / 2

d) Bloting : 1 / 2

e) Vomiting : 1 / 2

f) Nausea : 1 / 2

g) Anorexia : 1 / 2

h) Loss of appetite : 1 / 2

i) Dysphagia : 1 / 2

j) Belching : 1 / 2

k) GI bleeding $\rightarrow$ Haemetemesis

$\rightarrow$ Malena

1 $\Rightarrow$ Yes

2 $\Rightarrow$ No

DURATION OF SYMPTOMS : d / w / m / y (days/weeks/months/years)

ENDOSCOPIC FINDINGS : 1 / 2 / 3 / 4 / 5 / 6 / 7 / 8 / 9

	NAME	AGE	SEX	TOBACCO/SMOKING	ALCOHOL	NSAID's	DM	HYPERTENSION	UPPER ABDOMEN PAIN	HEART BURN	EARLY SATIETY	BLOATING	VOMITING	NAUSEA	BELCHING	LOSS OF APPETITE	DYSPHAGIA	LOSS OF WEIGHT	UPPER G.I BLEEDING	ANEMIA	DURATION OF SYMPTOMS	D / W / M / Y	NORMAL STUDY	ESOPHAGITIS	CA. ESOPHAGUS	GASTRITIS	GASTRIC ULCER	CA. STOMACH	DUODENAL ULCER	DUODENITIS	DUOD. STRICTURE/G.O.O
1	Seetha	54	F	2	2	2	2	1	1	1	2	2	2	1	1	2	2	2	2	2	3	m	2	2	2	1	2	2	2	2	2
2	Johnson	35	M	1	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2	20	d	1	2	2	2	2	2	2	2	2
3	Vasanthi	32	F	2	2	2	2	2	1	2	2	2	2	1	2	2	2	2	2	2	4	w	1	2	2	2	2	2	2	2	2
4	Vanjammal	45	F	2	2	2	2	2	1	1	1	2	1	1	1	2	2	2	2	2	2	m	2	1	2	1	2	2	2	2	2
5	Narasimma	47	M	1	1	2	2	2	2	1	2	2	2	1	2	2	2	2	2	2	6	m	1	2	2	2	2	2	2	2	2
6	Thenmozhi	18	F	2	2	2	2	2	1	1	2	2	1	1	2	2	2	2	2	2	6	w	1	2	2	2	2	2	2	2	2
7	Thenappan	62	M	1	2	2	1	1	2	1	2	2	2	1	1	2	2	2	2	1	8	m	2	1	2	2	2	2	2	2	2
8	Samsudeen	61	M	1	2	2	2	1	2	2	2	1	1	1	2	1	2	2	2	2	2	m	2	1	2	2	2	2	2	2	2
9	Balachandar	25	M	1	1	2	2	2	1	1	2	1	2	2	1	1	1	2	2	2	1	m	2	1	2	1	2	2	2	2	2
10	Kaladevi	28	F	2	2	2	2	2	1	1	2	2	2	1	2	2	2	2	2	2	3	w	1	2	2	2	2	2	2	2	2
11	vanaja	36	F	2	2	2	2	2	1	1	2	1	2	2	1	1	2	2	2	2	1	M	1	2	2	2	2	2	2	2	2
12	DEVARAJ	52	M	1	1	2	1	1	1	1	2	2	1	1	2	2	2	2	1	1	3	d	2	2	2	1	1	2	2	1	2
13	PREM	18	M	2	2	1	2	2	1	2	2	2	2	2	2	2	2	2	1	2	2	D	2	2	2	2	2	2	1	2	2
14	MANI	55	M	1	1	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2	1	W	1	2	2	2	2	2	2	2	2
15	KISHORE	20	M	1	2	2	2	2	1	2	2	2	2	2	2	1	2	2	2	2	3	W	2	2	2	2	2	2	1	2	2
16	NAVRATNRAJ	63	M	1	2	1	1	1	2	2	2	2	1	1	2	2	2	2	1	2	2	D	2	2	2	2	2	2	1	2	2
17	MUNUSAMY	31	M	1	2	2	2	2	1	1	2	2	2	2	2	2	2	2	2	2	4	M	1	2	2	2	2	2	2	2	2
18	DEVAPUTRI	66	F	2	2	2	1	2	1	2	2	1	1	1	1	2	2	2	2	2	5	w	2	2	2	2	2	2	2	1	2
19	PRAKASH	24	M	1	2	2	2	2	1	1	2	2	1	1	2	1	2	2	2	2	18	m	2	1	2	2	2	2	2	1	2
20	VADIVELU	35	M	2	2	2	2	2	1	1	2	2	2	2	2	2	2	2	2	2	4	m	1	2	2	2	2	2	2	2	2
21	SIVAPRASAD	30	M	2	2	1	2	2	1	1	2	2	2	2	2	2	2	2	2	2	6	w	2	1	2	2	2	2	2	2	2
22	PURUSHOTAM	62	M	1	2	1	1	2	2	2	2	2	1	2	2	2	2	2	1	2	2	d	2	2	2	2	2	2	1	2	2
23	MEHBOOB BEE	68	F	2	2	2	1	1	1	2	2	2	2	1	2	1	2	2	2	2	2	w	2	1	2	1	2	2	1	2	2
24	VENKATIAH	65	M	1	2	2	1	1	1	1	1	1	1	1	1	2	2	2	2	1	y	2	2	2	1	2	2	1	2	1	
25	SABIRA BEE	42	F	2	2	2	2	2	1	2	1	1	2	2	2	2	2	2	2	2	4	w	1	2	2	2	2	2	2	2	2
26	SRINIVASULU	52	M	2	2	2	2	2	1	1	2	2	2	2	2	2	2	2	2	2	1	w	2	2	2	1	2	2	2	2	2
27	BINU	18	F	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2	1	2	3	d	2	2	2	2	2	2	1	1	2
28	THANIKAVEL	28	M	1	2	2	2	2	2	2	2	2	2	2	2	1	2	1	2	2	3	m	1	2	2	2	2	2	2	2	2
29	MUNIAMMAL	68	F	2	2	1	1	1	1	1	1	2	2	2	1	1	2	2	2	2	2	m	2	1	2	2	2	2	2	2	2
30	VENKATESAN	30	M	2	2	2	2	2	1	1	2	2	2	1	2	2	2	2	2	2	3	w	2	2	2	1	2	2	2	2	2
31	BUVANESWARI	40	F	2	2	2	2	2	2	2	2	2	2	2	2	1	1	1	2	2	1	m	2	2	1	2	2	2	2	2	2
32	AROGYA MARY	50	F	2	2	2	2	2	1	2	1	2	2	1	2	2	2	2	2	1	6	m	1	2	2	2	2	2	2	2	2
33	PRAKASH	24	M	2	2	2	2	2	1	1	1	2	1	1	2	1	2	2	2	2	18	M	2	1	2	2	2	2	2	1	2
34	RAJARATNAM	72	M	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	Y	2	2	2	1	2	2	2	2	2
35	NANCY	21	F	2	2	2	2	2	2	2	2	1	1	2	2	2	2	2	2	2	1	M	1	2	2	2	2	2	2	2	2
36	SREEJA	25	F	2	2	1	2	2	1	1	2	2	2	2	2	2	2	2	2	2	3	W	1	2	2	2	2	2	2	2	2
37	THANGAMMAL	80	F	2	2	2	2	2	2	2	2	2	2	2	2	1	2	1	2	2	1	M	1	2	2	2	2	2	2	2	2
38	VEERIAH	78	M	1	2	1	1	1	1	1	2	2	2	1	2	2	2	2	2	2	4	M	1	2	2	2	2	2	2	2	2
39	REKHA	60	F	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2	1	1	Y	2	2	2	2	2	2	1	2	2
40	MALLIKARJUN	30	M	1	1	2	2	2	1	1	2	2	1	2	2	1	2	2	2	2	25	D	2	2	2	1	2	2	2	1	2
41	GEORGE	75	M	1	2	2	1	2	1	2	1	1	1	1	1	1	2	1	2	2	7	M	2	2	2	2	2	2	2	2	1
42	BUVANESWARI	30	F	2	2	2	2	2	2	1	2	2	1	1	2	1	2	2	2	2	2	Y	2	1	2	2	2	2	2	2	2
43	SAI KALA	40	F	2	2	2	2	2	1	2	2	2	1	1	2	2	2	2	2	2	2	W	1	2	2	2	2	2	2	2	2
44	VALLI	56	F	2	2	1	1	2	1	2	2	2	2	2	2	2	2	2	2	2	4	W	2	1	2	2	2	2	2	2	2
45	SENTHAMARAI	59	F	2	2	2	2	2	1	1	2	2	1	1	2	2	2	2	2	2	4	W	1	2	2	2	2	2	2	2	2
46	MARAGATHAM	50	F	2	2	2	2	2	1	2	2	2	1	2	2	1	2	2	2	2	6	W	2	2	2	1	2	2	2	2	2
47	GEETHA	48	F	2	2	2	2	1	1	1	2	2	2	1	2	1	2	2	2	2	3	M	1	2	2	2	2	2	2	2	2
48	ARUMUGAM	76	M	1	2	2	2	1	2	2	2	2	2	1	2	2	2	2	2	2	3	W	2	1	2	2	2	2	2	2	2
49	MALATHY	30	F	2	2	2	2	2	1	1	2	2	2	2	2	2	2	2	2	2	5	M	2	1	2	2	2	2	2	2	2
50	RAMESH	26	M	1	2	2	2	2	2	1	1	2	2	1	2	2	2	2	2	2	6	W	1	2	2	2	2	2	2	2	2

51	JOSEPH	41	M	1	2	2	2	2	1	1	2	2	2	2	2	2	2	2	2	2	1	M	2	1	2	2	2	2	2	2	2
52	SHANTAMMA	59	F	2	2	2	2	2	2	1	1	2	2	1	1	2	2	2	2	2	6	M	1	2	2	2	2	2	2	2	2
53	LAXMI	48	F	2	2	2	2	2	2	2	1	1	2	1	1	2	2	2	2	2	4	M	1	2	2	2	2	2	2	2	2
54	DANIEL	81	M	2	2	2	1	2	2	2	2	2	2	2	2	1	2	2	2	8	M	1	2	2	2	2	2	2	2	2	2
55	ESHOK	24	M	2	2	2	2	2	1	2	2	2	1	2	2	2	1	2	2	2	3	M	2	2	2	2	2	2	1	2	2
56	AMBALAMMAL	65	F	2	2	1	1	1	1	1	1	1	2	1	1	2	2	2	2	2	6	W	2	2	2	1	1	2	2	2	2
57	RATHI	51	F	2	2	2	2	2	2	2	2	1	2	2	1	1	2	2	2	2	25	D	1	2	2	2	2	2	2	2	2
58	JOTHY	25	F	2	2	2	2	2	1	2	2	2	2	2	2	1	2	2	2	2	3	W	2	2	2	2	2	2	2	1	2
59	GANESAN	32	M	1	1	2	2	2	1	1	2	2	2	1	2	1	2	2	2	2	1	W	2	2	2	2	2	2	2	1	2
60	JEYALAXMI	55	F	2	2	2	2	2	2	1	2	2	1	1	2	1	2	2	2	2	5	M	2	1	2	2	2	2	2	2	2
61	PRABHAVATHY	21	F	2	2	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	6	W	1	2	2	2	2	2	2	2	2
62	CHOKAMMAL	80	F	2	2	2	1	2	2	2	1	2	2	1	2	2	2	2	2	2	6	M	1	2	2	2	2	2	2	2	2
63	ISMAIL	24	M	1	2	1	2	1	1	1	2	2	2	2	2	2	2	2	2	2	4	W	2	2	2	1	2	2	2	2	2
64	SATISH	29	M	2	2	2	2	2	1	2	2	2	1	1	2	2	2	2	2	2	3	W	1	2	2	2	2	2	2	2	2
65	ANAND RAO	57	M	1	1	2	1	2	2	1	1	2	2	2	1	1	2	2	2	2	1	Y	2	2	2	1	2	2	2	2	2
66	ANANDHI	37	F	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	W	1	2	2	2	2	2	2	2	2
67	RAJESWARI	33	F	2	2	2	2	2	1	2	2	2	2	2	2	1	2	2	2	2	2	M	2	1	2	2	2	2	2	2	2
68	KANNIAMMAL	82	F	2	2	2	1	2	2	1	2	2	2	2	1	1	2	2	2	2	7	M	1	2	2	2	2	2	2	2	2
69	NAGAMMAL	70	F	2	2	1	1	2	1	2	2	2	2	2	1	1	2	2	2	1	1	Y	2	2	2	1	2	2	2	2	2
70	PANNERSELVAM	50	M	1	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2	6	W	1	2	2	2	2	2	2	2	2
71	PAARVATHY	60	F	2	2	1	2	1	2	1	2	2	2	2	2	1	2	2	2	1	20	D	2	2	2	1	2	2	2	2	2
72	MUNUSAMY	45	M	1	2	2	2	2	1	1	2	2	2	1	1	2	2	2	2	2	6	W	2	2	2	2	2	2	1	2	2
73	GEORGE	65	M	1	1	2	2	2	2	2	2	1	2	2	1	2	2	2	2	3	W	2	2	2	1	2	2	2	2	2	2
74	ANANDHAN	55	M	1	1	2	2	1	2	2	1	2	2	1	1	2	2	2	2	2	4	M	1	2	2	2	2	2	2	2	2
75	PREMA	44	F	2	2	2	2	2	1	2	2	1	2	2	2	2	2	2	2	2	2	M	1	2	2	2	2	2	2	2	2
76	GEETHA	41	F	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2	25	D	1	2	2	2	2	2	2	2	2	2
77	GEJALAXMI	23	F	2	2	2	2	2	1	2	2	2	1	2	2	2	2	2	2	2	1	Y	1	2	2	2	2	2	2	2	2
78	SIMON FRANCIS	45	M	1	2	2	2	2	1	1	1	2	2	2	2	2	2	2	2	2	7	W	2	2	2	2	2	2	2	1	2
79	VINYAGAM	60	M	1	1	2	1	2	1	1	2	1	2	2	2	1	2	2	2	2	3	W	2	1	2	2	2	2	2	2	2
80	SHANKAR PETEF	49	M	2	2	2	2	2	1	1	2	2	2	1	2	2	2	2	2	2	25	D	2	2	2	1	2	2	2	2	2
81	CHITRA	25	F	2	2	2	2	2	2	1	1	1	2	2	2	2	2	2	2	2	2	M	1	2	2	2	2	2	2	2	2
82	RAJALAXMI	23	F	2	2	2	2	2	1	1	2	1	2	1	2	2	2	2	2	2	1	M	1	2	2	2	2	2	2	2	2
83	RAMEEJA	27	F	2	2	2	2	2	1	1	2	2	1	2	2	2	1	2	2	2	2	Y	1	2	2	2	2	2	2	2	2
84	KANAGARATNAM	41	F	2	2	2	2	2	1	2	1	2	2	1	2	2	2	2	2	2	5	W	2	2	2	1	2	2	2	2	2
85	KANNAN	29	M	1	2	2	2	2	1	1	2	1	2	2	1	2	2	2	2	2	4	W	2	2	2	1	2	2	2	1	2
86	JASMINE VIJI	35	F	2	2	2	2	2	1	2	2	2	1	1	2	1	2	2	2	2	25	D	1	2	2	2	2	2	2	2	2
87	SHAHIDA	37	F	2	2	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	W	2	2	2	1	2	2	2	2	2
88	MANOHAR	26	M	2	2	2	2	2	1	2	2	2	2	2	2	2	1	2	2	2	4	Y	1	2	2	2	2	2	2	2	2
89	KALAVATHY	23	F	2	2	2	2	2	1	1	2	2	1	1	2	2	2	2	2	2	6	W	2	1	2	2	2	2	2	2	2
90	VELAYUDHRAJ	22	M	1	1	2	2	2	2	1	2	2	1	1	2	2	2	2	2	2	3	M	2	2	2	1	2	2	2	2	2
91	DHANALAXMI	16	F	2	2	2	2	2	1	2	2	2	2	1	2	1	2	2	2	2	1	Y	1	2	2	2	2	2	2	2	2
92	MANIKAM	80	M	2	2	2	2	2	2	1	2	1	2	2	1	1	2	2	2	2	1	M	1	2	2	2	2	2	2	2	2
93	DEENADAYAL	34	M	1	2	1	2	2	1	1	2	2	1	2	2	2	2	2	2	2	25	D	1	2	2	2	2	2	2	2	2
94	MUNEESWARI	36	F	2	2	2	2	2	1	2	2	2	2	2	1	2	2	2	2	2	7	M	2	1	2	1	2	2	2	2	2
95	GUNASEKAR	54	M	1	1	2	2	2	1	1	2	1	2	1	2	2	2	2	2	2	1	Y	2	2	2	1	2	2	2	2	2
96	SULOCHANA	34	F	2	2	2	2	2	1	2	2	2	1	2	2	1	1	2	2	2	3	M	2	1	2	1	2	2	2	2	2
97	MALADRI	42	M	1	2	2	2	2	1	2	1	1	2	2	2	1	2	2	2	2	1	M	1	2	2	2	2	2	2	2	2
98	ARUMUGAM	76	M	1	2	2	1	2	1	2	2	1	1	1	2	2	2	2	2	2	3	M	2	2	2	1	2	2	2	1	2
99	ALICE	59	F	2	2	2	2	2	1	2	2	2	2	1	2	1	2	2	2	2	6	M	1	2	2	2	2	2	2	2	2
100	DESINGH	54	M	2	2	1	2	1	1	2	2	2	2	2	2	2	2	2	2	2	1	M	2	2	2	2	2	2	2	1	2
101	VASUDEVAN	45	M	1	1	1	2	2	1	1	2	2	2	1	2	2	2	2	2	2	2	W	2	1	2	1	2	2	2	2	2
102	BASKAR	38	M	1	2	2	2	2	1	1	2	2	2	2	2	2	2	2	2	2	1	M	2	1	2	1	2	2	2	2	2
103	ARUMUGAM	31	M	1	2	2	2	2	1	2	2	2	1	1	2	2	2	2	2	2	2	M	2	2	2	1	2	2	2	2	2
104	VIJAYANATHAN	30	M	2	2	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2	6	W	1	2	2	2	2	2	2	2	2
105	MANOHAR	38	M	1	2	2	2	2	2	1	2	2	2	1	2	2	2	2	2	2	3	M	2	2	2	1	2	2	1	2	2
106	NAGAMMAL	76	F	2	2	2	1	1	2	1	1	1	2	2	1	2	2	2	2	1	1	Y	1	2	2	2	2	2	2	2	2
107	LATHA	47	F	2	2	1	2	2	1	1	2	2	1	2	2	2	2	2	2	2	5	M	1	2	2	2	2	2	2	2	2
108	LOGANATHAN	72	M	2	2	2	2	2	2	1	2	2	1	1	2	2	2	2	2	2	3	M	2	2	2	1	2	2	2	2	2
109	KARTHIKEYAN	16	M	2	2	2	2	2	1	1	2	2	2	1	2	2	2	2	2	2	2	W	2	2	2	1	2	2	2	1	2
110	SEKAR	46	M	1	2	2	2	2	1	1	2	2	2	2	2	1	2	2	2	2	3	W	2	1	2	2	2	2	2	2	2

111	VIOLET RANI	53	F	2	2	1	1	1	1	2	2	1	2	1	2	1	2	2	2	2	25	D	1	2	2	2	2	2	2	2	2
112	RAJ KUMAR	35	M	1	1	2	2	2	1	1	2	2	2	2	2	2	2	2	2	2	2	W	2	2	2	1	2	2	2	2	2
113	SATYA MORTHY	34	M	2	2	1	2	2	1	2	2	1	2	2	2	2	2	2	2	2	1	W	1	2	2	2	2	2	2	2	2
114	TERRENCE	23	M	2	2	2	2	2	1	1	2	2	1	2	2	2	2	2	2	2	1	M	2	2	2	1	2	2	2	2	2
115	MEENAKSHI	67	F	2	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2	1	3	W	1	2	2	2	2	2	2	2	2
116	SAROJINI	65	F	2	2	1	2	2	1	2	2	2	1	1	2	2	2	2	2	2	4	W	1	2	2	2	2	2	2	2	2
117	INDIRA	57	F	2	2	2	2	2	1	2	2	2	1	2	2	1	2	2	2	2	3	Y	1	2	2	2	2	2	2	2	2
118	SHYAMALA	47	F	2	2	1	2	2	1	2	2	2	1	2	2	1	1	2	2	2	1	M	2	2	2	1	2	2	2	2	2
119	CHANDRAN	28	M	1	2	2	2	2	1	1	2	2	1	2	2	2	2	2	2	2	4	Y	2	2	2	2	2	2	2	1	2
120	ABINANDHAN	30	M	2	2	2	2	2	1	1	2	2	1	1	2	2	2	2	2	2	1	W	2	2	2	2	2	2	2	1	2
121	ROSARY DASAN	44	M	1	2	1	1	2	1	2	2	2	2	1	2	2	2	2	2	2	2	M	2	2	2	1	2	2	2	2	2
122	KUMARASAMY	52	M	2	2	2	2	2	1	1	2	1	2	2	2	1	2	2	2	2	6	M	1	2	2	2	2	2	2	2	2
123	LAXMI	26	F	2	2	2	2	2	1	2	2	2	1	1	2	2	2	2	2	2	2	Y	1	2	2	2	2	2	2	2	2
124	SUDHA	22	F	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	4	W	1	2	2	2	2	2	2	2	2
125	JAMES	76	M	2	2	1	2	2	2	1	2	2	2	2	2	2	2	1	2	6	W	1	2	2	2	2	2	2	2	2	2
126	KADAR BAHADUI	31	M	1	1	2	2	2	1	1	2	1	1	1	2	2	2	2	2	2	2	M	1	2	2	2	2	2	2	2	2
127	LOGANATHAN	72	M	2	2	2	2	2	1	2	2	2	1	2	2	1	1	2	2	2	6	M	2	1	2	2	2	2	2	2	2
128	MAN SINGH	25	M	2	2	1	2	2	1	1	2	2	2	1	2	1	2	2	2	2	2	M	2	2	2	1	2	2	2	1	2
129	SHANTI	30	F	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	1	M	1	2	2	2	2	2	2	2	2
130	KONDIAH	39	M	1	2	2	1	2	2	2	2	1	1	1	1	1	2	2	2	2	1	W	2	2	2	1	2	2	2	2	2
131	SIVALINGAM	48	M	1	1	2	1	2	1	1	2	2	2	1	2	2	2	2	2	2	1	Y	2	1	2	2	2	2	2	2	2
132	PRAMILA	50	F	2	2	2	2	2	2	1	1	1	2	2	2	1	2	2	2	2	6	M	1	2	2	2	2	2	2	2	2
133	MANOHAR	45	M	1	2	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2	D	2	2	2	1	2	2	2	2	2
134	PAPATHIAMMAL	75	F	2	2	2	2	2	1	2	2	2	1	2	2	1	2	2	2	2	2	W	1	2	2	2	2	2	2	2	2
135	SUBRAMANIAM	46	M	2	2	2	2	2	1	1	1	1	2	2	2	2	2	2	2	2	1	W	2	2	2	1	2	2	2	2	2
136	ZULAI KABI	53	F	2	2	2	2	2	1	2	2	2	2	2	2	1	1	2	2	2	6	M	1	2	2	2	2	2	2	2	2
137	KUMAR	30	M	1	2	2	2	2	1	1	1	1	1	1	1	2	2	2	2	2	2	M	2	1	2	2	2	2	2	2	2
138	ELSAMMA	36	F	2	2	2	2	2	1	2	1	1	2	1	2	1	2	1	2	2	4	M	2	2	2	2	2	1	2	2	2
139	JOEL FAITH	22	M	2	2	2	2	2	1	1	2	2	2	2	2	1	2	2	2	2	5	M	2	2	2	1	2	2	1	2	2
140	PRAKASAM	45	M	1	2	1	2	2	1	2	2	2	1	1	2	2	2	2	2	2	4	W	2	2	2	1	2	2	2	1	2
141	MURUGAN	48	M	2	2	1	2	2	1	2	2	2	2	1	2	2	2	2	2	2	1	Y	2	2	2	2	2	2	1	2	2
142	VISWANATHAN	55	M	1	1	2	2	2	1	2	2	2	1	2	2	1	2	2	2	2	5	M	2	1	2	2	2	2	2	2	2
143	RAVI	35	M	1	2	2	2	2	1	1	2	2	2	2	2	2	2	2	2	2	1	M	2	2	2	1	2	2	2	2	2
144	MAHESWARI	30	F	2	2	2	2	2	1	1	2	2	2	1	2	2	2	2	2	2	1	Y	2	1	2	2	2	2	2	2	2
145	DOSS	74	M	2	2	2	2	1	1	2	2	2	2	2	2	2	2	2	2	1	3	M	2	2	2	2	2	2	1	1	2
146	SUNDARARAJ	59	M	2	2	1	2	2	2	1	1	1	2	2	1	2	2	2	2	2	6	W	2	2	2	1	2	2	2	1	2
147	THENMOZHI	35	F	2	2	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2	5	M	2	1	2	2	2	2	2	1	2
148	UMA MAHESWAF	38	F	2	2	2	2	2	1	1	2	2	2	2	2	2	2	2	2	2	1	W	1	2	2	2	2	2	2	2	2
149	BALASUBRAMAN	39	M	1	2	2	2	2	2	1	2	2	2	2	2	1	2	2	2	2	2	M	2	2	2	1	2	2	2	1	2
150	SUBADRA DEVI	61	F	2	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2	8	M	1	2	2	2	2	2	2	2	2
151	JOEL	22	M	2	2	2	2	2	1	2	2	2	2	1	2	2	2	2	1	2	2	M	2	2	2	1	2	2	2	2	2
152	SAKKUBAI	60	F	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	3	M	1	2	2	2	2	2	2	2	2
153	KIRUBAVTHY	65	F	2	2	2	2	2	2	2	1	1	2	1	1	1	2	2	2	2	8	W	1	2	2	2	2	2	2	2	2
154	BOOPALAN	48	M	1	1	2	2	2	1	2	2	2	1	1	2	2	2	2	2	2	4	M	2	2	2	2	2	2	2	1	1
155	LINGESWARAN	53	M	1	2	2	2	1	1	1	2	2	2	1	2	2	2	2	2	2	20	D	2	1	2	2	2	2	2	2	2
156	DEENADAYAL	39	M	2	2	2	2	2	1	1	2	2	1	1	2	2	2	1	2	1	6	M	2	2	2	1	2	2	2	2	2
157	ABRAHAM	44	M	1	1	2	2	2	1	2	2	2	1	2	2	2	2	2	2	2	20	D	2	2	2	1	2	2	2	2	2
158	ANNADURAI	45	M	1	2	2	2	2	1	1	2	2	1	2	2	2	2	2	2	2	3	W	1	2	2	2	2	2	2	2	2
159	AMUDHA	35	F	2	2	1	2	2	1	1	2	2	2	1	2	2	2	2	2	2	1	W	2	2	2	1	2	2	2	2	2
160	BALAJI	22	M	2	2	2	2	2	1	2	2	2	1	1	2	1	2	2	2	2	1	Y	2	2	2	1	2	2	2	1	2
161	THANGARAJ	60	M	1	2	1	2	2	1	1	2	2	1	1	2	2	2	2	2	2	4	M	2	1	2	2	2	2	2	2	2
162	YADAV	30	M	1	1	2	2	2	1	1	2	1	1	1	2	1	2	2	2	2	6	M	2	1	2	2	2	2	2	2	2
163	ANSAR BEE	40	F	2	2	2	2	1	1	1	2	2	1	1	2	1	2	2	2	1	2	W	2	2	2	1	2	2	2	2	2
164	MUTHU PANDI	23	M	1	2	2	2	2	1	1	2	2	2	2	2	2	2	2	2	2	2	M	2	2	2	1	2	2	2	2	2
165	TAMIMA	28	F	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	1	Y	1	2	2	2	2	2	2	2	2
166	SUMATHI	40	F	2	2	2	2	2	1	1	2	2	2	2	1	2	2	2	2	2	2	Y	1	2	2	2	2	2	2	2	2
167	PADMANABAN	55	M	1	1	2	2	2	2	2	2	2	1	1	1	1	2	2	2	2	8	M	2	1	2	2	2	2	2	2	2
168	PORSELVAM	31	M	1	1	2	2	2	1	1	2	2	1	1	2	2	2	2	2	2	2	W	2	1	2	1	2	2	2	2	2
169	BHARATHI	17	F	2	2	2	2	2	1	2	2	2	1	1	2	2	2	2	2	2	3	M	2	2	2	1	2	2	2	2	2
170	KASTHURI	42	F	2	2	2	2	2	1	2	2	2	1	1	2	2	2	1	2	2	2	M	1	2	2	2	2	2	2	2	2

171	RAJAN	32	M	1	1	1	2	2	1	1	2	2	2	1	2	2	2	2	2	2	1	Y	2	1	2	2	2	2	2	1	2
172	SHANTHI	43	F	2	2	2	2	2	2	1	1	1	1	1	2	1	2	2	2	2	2	M	2	1	2	2	2	2	2	2	2
173	NAGAVENI	32	F	2	2	2	2	2	2	1	2	1	2	2	1	2	2	2	2	2	6	W	1	2	2	2	2	2	2	2	2
174	KUMARAN	22	M	1	2	1	2	2	1	2	2	1	1	2	2	2	2	2	2	2	2	M	2	1	2	2	2	2	2	1	2
175	RAJAMANIKKAM	73	M	2	2	2	1	1	1	2	2	2	1	1	2	2	2	2	2	2	6	M	2	2	2	1	2	2	2	2	2
176	CHANDRA	60	F	2	2	2	2	1	2	2	2	2	2	2	2	1	1	1	2	1	2	M	2	2	1	2	2	2	2	2	2
177	SULTANA BEGUM	34	F	2	2	2	2	2	1	1	2	1	2	1	2	1	2	2	2	2	1	Y	2	1	2	1	2	2	2	2	2
178	RANI	27	F	2	2	2	2	2	1	2	1	2	2	2	2	2	2	2	2	2	25	D	2	2	2	1	2	2	2	2	2
179	RAVI SANKAR	38	M	1	1	2	2	2	1	2	2	2	1	1	2	2	2	2	2	2	1	W	2	2	2	1	2	2	2	2	2
180	JEYAMMAL	65	F	2	2	2	1	1	2	2	2	2	2	2	2	2	2	2	1	2	5	M	2	2	2	1	2	2	2	2	2
181	VISALATCHI	58	F	2	2	2	1	1	1	2	2	1	1	1	2	2	2	2	2	2	1	M	1	2	2	2	2	2	2	2	2
182	PARIMALA	57	F	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	3	M	2	1	2	2	2	2	2	2	2
183	VASUGI	51	F	2	2	1	2	1	1	1	2	1	2	2	2	2	2	2	2	2	2	W	2	2	2	1	2	2	2	2	2
184	VASANTHA	45	F	2	2	1	2	2	1	1	2	2	2	2	2	2	2	2	2	2	1	W	2	1	2	2	2	2	2	2	2
185	ABDUL LATIF	85	M	2	2	2	2	2	2	2	1	1	1	1	2	1	1	1	2	2	1	M	2	2	2	2	2	1	2	2	2
186	MANJULA	27	F	2	2	2	2	2	1	1	1	2	2	1	2	1	2	2	2	2	3	W	1	2	2	2	2	2	2	2	2
187	MOHANA	41	F	2	2	2	2	2	1	2	2	2	2	1	2	1	2	2	2	1	6	M	1	2	2	2	2	2	2	2	2
188	KALPANA	21	F	2	2	2	2	2	1	2	2	2	1	2	2	2	2	2	2	2	2	Y	2	2	2	1	2	2	2	2	2
189	MALLIKA	45	F	2	2	2	2	2	1	1	2	2	2	2	2	2	2	2	2	2	6	M	2	2	2	1	2	2	2	2	2
190	ESTHER	62	F	2	2	2	2	2	1	2	2	2	2	2	2	1	2	2	2	2	3	M	1	2	2	2	2	2	2	2	2
191	AMALORPARANI	31	F	2	2	1	2	2	1	1	2	2	2	1	2	2	2	2	2	2	1	W	2	1	2	2	2	2	2	2	2
192	SUNDARAMBAL	60	F	2	2	1	2	2	2	1	2	2	2	2	2	2	2	2	2	1	20	D	2	2	2	1	2	2	2	2	2
193	PAPPAMMAL	52	F	2	2	2	2	1	2	1	2	2	2	2	1	2	2	2	2	2	2	M	1	2	2	2	2	2	2	2	2
194	JAMES	38	M	1	2	1	2	2	1	1	2	2	2	1	2	1	2	2	2	2	3	M	2	1	2	1	2	2	2	2	2
195	MUNIAMMAL	43	F	2	2	2	2	2	2	2	2	2	1	1	2	2	2	2	2	2	1	M	1	2	2	2	2	2	2	2	2
196	SUSEELA	41	F	2	2	1	2	2	1	1	2	2	2	2	2	2	2	2	2	2	6	W	2	2	2	2	2	2	2	1	2
197	VINOD KUMAR	17	M	2	2	2	2	2	1	1	2	1	2	2	1	2	2	2	2	2	8	M	2	2	2	1	2	2	2	2	2
198	PREMAVATHY	41	F	2	2	2	2	2	1	1	2	2	1	1	2	2	2	2	2	2	3	W	2	1	2	2	2	2	2	2	2
199	CHANDRA	60	F	2	2	2	1	1	2	2	2	2	1	1	2	1	1	2	2	2	2	M	1	2	2	2	2	2	2	2	2
200	UMA	35	F	2	2	2	2	2	1	2	2	1	1	2	1	2	2	2	2	2	3	W	2	1	2	2	2	2	2	2	2
201	BHAVANI	42	F	2	2	1	2	2	1	1	2	2	2	2	2	1	2	2	2	2	2	M	2	1	2	2	2	2	2	2	2
202	SELVI	37	F	2	2	1	2	2	1	2	1	1	1	1	1	2	2	2	2	2	6	M	2	1	2	2	2	2		2	2
203	VIMALA	28	F	2	2	2	2	2	1	2	2	2	1	2	2	2	2	2	2	2	20	D	2	1	2	2	2	2	2	2	2
204	RAJESWARI	17	F	2	2	2	2	2	1	1	2	2	2	2	2	2	2	2	2	2	3	M	2	1	2	2	2	2	2	2	2
205	MADAN KUMAR	23	M	1	2	2	2	2	1	1	2	2	2	2	2	1	2	2	2	2	3	W	2	1	2	1	2	2	2	2	2
206	KOMALAVALLI	31	F	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	25	D	1	2	2	2	2	2	2	2	2
207	AWDESH KUMAR	50	M	1	1	1	2	2	1	1	2	1	2	2	2	2	2	2	2	2	2	W	1	2	2	2	2	2	2	2	2
208	GOPINATH	63	M	1	2	2	2	2	1	2	2	1	2	1	1	2	2	2	2	2	4	M	2	2	2	2	2	2	2	1	2
209	RAMESH	20	M	1	2	2	2	2	1	1	2	2	2	2	2	1	2	2	2	2	3	M	2	2	2	2	2	2	1	2	2
210	SARASWATHY	48	F	2	2	2	2	2	1	2	2	2	1	2	2	2	2	2	2	2	2	W	2	2	2	1	2	2	2	2	2
211	ANKIAH	25	M	1	1	2	2	2	2	2	1	1	1	2	2	2	2	2	2	2	3	M	1	2	2	2	2	2	2	2	2
212	SURESH	32	M	1	2	2	2	2	1	2	2	2	2	1	2	2	2	2	2	2	8	M	2	2	2	2	2	2	1	2	1
213	PADMAVATHY	53	F	2	2	1	2	2	2	2	2	2	1	2	2	2	2	2	1	1	1	W	2	2	2	2	2	2	1	1	2
214	SRINIVASAN	34	M	2	1	2	2	2	1	1	2	2	2	2	2	2	2	2	2	2	3	Y	2	2	2	1	2	2	2	2	2
215	VIJAYAKUMARI	52	F	2	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2	5	M	2	1	2	2	2	2	2	2	2
216	SAI BASKAR	33	M	1	1	2	2	2	1	1	2	2	2	2	2	2	2	2	2	2	1	M	2	1	2	2	2	2	2	2	2
217	MARGRET	50	F	2	2	1	2	1	1	1	2	2	1	1	2	2	2	2	2	2	6	W	2	2	2	2	2	2	2	1	2
218	AKBAR PASHA	25	M	1	1	2	2	2	1	1	2	2	2	1	1	1	2	2	2	2	3	W	2	2	2	2	2	2	2	1	2
219	BAALIAH	29	M	2	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2	1	Y	2	1	2	2	2	2	2	2	2
220	KALAVATHY	46	F	2	2	1	2	2	1	1	2	2	2	2	2	2	1	2	2	1	5	Y	2	1	2	2	2	2	2	2	2
221	BAABU	70	M	1	2	2	1	2	2	2	2	2	1	1	2	1	1	2	2	2	25	D	2	1	2	2	2	2	2	2	2
222	DURAI KANNU	69	M	1	2	2	2	2	2	1	1	1	2	2	1	2	2	2	2	2	4	M	2	2	2	1	2	2	2	2	2

223	NEELAMMA	66	F	2	2	2	2	2	1	1	2	2	2	2	2	2	2	2	2	2	1	Y	1	2	2	2	2	2	2	2	2
224	VENKIAH	35	M	1	1	1	2	2	1	1	2	1	2	2	2	1	2	2	2	2	2	M	2	2	2	2	1	2	2	2	2
225	SANTHANAM	55	M	2	2	1	2	2	2	2	2	2	1	2	2	2	2	2	1	2	3	W	2	2	2	1	2	2	2	2	2
226	MANIKANDAN	15	M	2	2	2	2	2	1	2	2	2	1	1	2	2	2	2	2	2	1	W	2	2	2	1	2	2	2	2	2
227	KUMUDHAM	74	F	2	2	2	1	2	1	2	2	2	2	2	2	2	2	2	2	2	4	M	1	2	2	2	2	2	2	2	2
228	ARASUPATHU	35	M	2	2	2	2	2	2	1	2	2	2	1	2	2	2	2	2	2	20	D	1	2	2	2	2	2	2	2	2
229	SAROJAMMA	62	F	2	2	2	1	1	2	2	2	2	2	1	2	1	2	2	2	2	1	M	2	2	2	1	2	2	2	2	2
230	PUSHPA	55	F	2	2	2	1	2	1	2	1	2	1	1	2	1	2	2	2	2	6	M	1	2	2	2	2	2	2	2	2
231	INDHUMATHY	25	F	2	2	2	2	2	1	1	2	2	2	2	2	2	2	2	2	2	6	W	2	1	2	2	2	2	2	2	2
232	RAJESWARI	50	F	2	2	1	2	2	1	2	1	2	2	2	2	1	2	2	2	2	2	M	2	2	2	1	2	2	2	2	2
233	DILLIBAI	42	F	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	3	M	2	2	2	2	2	2	1	2	1
234	ELAVARASI	40	F	2	2	2	2	2	1	2	2	2	1	1	2	2	2	2	2	2	3	M	2	2	2	1	2	2	2	2	2
235	PREMA	44	F	2	2	2	2	2	1	2	2	2	1	2	2	2	2	2	2	2	1	Y	2	1	2	2	2	2	2	2	2
236	SREEDEVI	26	F	2	2	1	2	2	1	2	1	2	1	1	1	2	2	2	2	2	1	M	2	1	2	2	2	2	2	2	2
237	NEETHU	23	F	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	4	W	1	2	2	2	2	2	2	2	2
238	SEBASTIN	38	M	1	1	2	2	2	1	1	2	2	2	1	2	2	2	2	2	2	4	M	2	2	2	2	1	2	2	2	2
239	ASHOK	23	M	1	2	2	2	2	1	1	2	2	2	1	1	2	2	2	2	2	2	W	2	1	2	2	1	2	2	2	2
240	KASTHURI	45	F	2	2	2	2	2	1	1	2	2	2	2	2	2	2	2	1	2	3	D	1	2	2	2	2	2	2	2	2
241	WILSON	36	M	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	6	M	2	2	2	2	2	2	1	2	2
242	BHIMARAJ	40	M	1	1	2	2	2	1	1	2	2	2	2	2	1	2	2	2	2	3	Y	2	1	2	2	2	2	2	2	2
243	UMA RANI	17	F	2	2	1	2	2	1	1	2	2	2	1	2	1	2	2	2	2	1	W	2	2	2	1	2	2	2	2	2
244	LOGAMMAL	66	F	2	2	1	1	1	1	2	2	1	1	2	2	1	2	2	2	2	1	M	2	1	2	2	2	2	2	2	2
245	BASKARAN	42	M	1	2	1	2	2	2	1	2	2	2	1	2	2	2	2	2	2	20	D	2	2	2	1	2	2	2	2	2
246	LOGANAYAGI	60	F	2	2	2	1	2	1	2	2	2	1	1	2	1	2	2	2	2	2	M	2	1	2	2	2	2	2	2	2
247	SHANTHI	36	F	2	2	2	2	2	1	2	2	2	2	2	2	1	2	2	2	2	6	M	2	1	2	2	2	2	2	2	2
248	INDRA MARY	54	F	2	2	1	2	2	2	2	2	2	1	1	2	2	1	1	2	2	5	M	2	2	2	1	2	2	2	2	2
249	MANGALAM	52	F	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2	10	M	1	2	2	2	2	2	2	2	2	2
250	PATTAMMAL	65	F	2	2	2	2	2	2	2	1	2	1	2	2	1	2	1	2	1	3	M	2	2	2	2	2	1	2	2	2
251	SARADHA	65	F	2	2	1	2	2	2	2	2	2	2	2	2	2	2	1	2	3	D	2	2	2	2	2	2	1	2	2	2
252	JOHN FRANKLIN	31	M	1	1	2	2	2	2	1	2	2	2	2	2	1	2	2	2	2	1	M	1	2	2	2	2	2	2	2	2
253	MUNUSAMY	39	M	1	1	1	2	2	1	2	2	2	1	1	2	2	1	2	2	2	25	D	2	1	2	2	2	2	2	2	2
254	SAKTHI RANI	33	F	2	2	1	2	2	1	1	2	2	1	1	2	1	2	2	2	2	2	W	2	2	2	1	2	2	2	2	2
255	PRAVEEN	18	M	1	2	2	2	2	1	1	2	2	2	2	2	2	2	2	2	2	4	M	2	1	2	2	2	2	2	1	2
256	MUKILAN	25	M	2	2	2	2	2	2	1	2	2	1	1	2	2	2	2	2	2	2	Y	2	1	2	2	2	2	2	2	2
257	MUNIAMMAL	65	F	2	2	2	2	2	1	1	2	2	2	2	2	2	2	2	2	2	1	Y	2	1	2	2	2	2	2	2	2
258	ESWARI	25	F	2	2	1	2	2	1	1	2	2	2	1	2	2	2	2	1	2	2	D	2	2	2	1	2	2	2	2	2
259	SHANMUGAM	25	M	1	1	2	2	2	2	1	1	1	2	2	1	2	2	2	2	2	3	M	2	2	2	1	2	2	2	2	2
260	RAJA PILLAI	39	M	1	2	2	2	2	2	2	1	1	1	1	1	1	2	2	2	2	2	M	2	2	2	2	2	2	2	2	1
261	SHANTAKUMARI	40	F	2	2	2	2	2	2	1	1	2	2	2	2	2	2	2	2	2	6	D	1	2	2	2	2	2	2	2	2
262	PETER JOHN	38	M	1	2	2	2	2	2	2	1	2	2	1	2	1	2	2	2	2	4	M	2	1	2	2	2	2	2	2	2
263	JEEVANESAN	61	M	1	2	2	2	2	2	2	2	2	2	2	2	1	2	1	1	1	1	W	2	2	2	2	2	2	1	2	2
264	PALANI	65	M	1	1	1	2	2	2	2	2	2	1	1	2	2	2	2	2	2	1	M	1	2	2	2	2	2	2	2	2
265	PERIASAMY	68	M	1	2	2	1	2	1	1	2	1	1	2	2	1	2	1	2	1	6	M	2	2	2	2	2	2	1	2	1
266	KAMILLA	54	F	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2	3	M	1	2	2	2	2	2	2	2	2	2
267	GHOUSE BEE	50	F	2	2	1	2	2	1	2	1	2	1	1	1	1	2	2	2	2	3	M	2	1	2	1	2	2	2	2	2
268	SUSEELA	65	F	2	2	2	1	1	2	1	2	2	2	1	2	1	2	2	2	2	3	W	2	1	2	2	2	2	2	2	2
269	DURAI	60	M	2	1	2	2	1	2	1	2	2	2	2	2	2	2	2	1	2	4	W	2	2	2	2	1	2	2	1	2
270	PARTHIBAN	40	M	1	1	2	2	2	1	2	2	2	2	1	2	1	2	2	2	2	5	W	2	2	2	1	2	2	2	2	2
271	RATHINAM	78	M	2	1	2	1	1	2	1	2	2	1	2	2	2	2	2	1	1	2	M	2	1	2	1	2	2	2	2	2
272	NARMADHA	26	F	2	2	2	2	2	2	1	2	2	2	1	2	1	2	2	2	2	6	M	1	2	2	2	2	2	2	2	2
273	GANIAN	69	M	1	1	2	2	2	1	2	2	2	1	2	2	2	2	2	2	1	4	W	2	1	2	2	2	2	2	2	2
274	CHANDRAVADAN	64	M	2	2	2	2	2	2	2	2	2	1	2	2	1	1	1	2	2	10	M	2	2	1	2	2	2	2	2	2
275	MUNIAMMA	65	F	2	2	1	2	2	1	2	2	2	2	2	1	2	2	2	2	2	6	M	1	2	2	2	2	2	2	2	2
276	MERCY	40	F	2	2	2	2	2	1	2	2	2	1	2	2	2	2	2	2	2	2	W	1	2	2	2	2	2	2	2	2
277	PRABHU	20	M	1	2	2	2	2	1	1	1	1	2	2	1	1	2	2	2	2	5	M	2	1	2	2	2	2	1	1	2
278	DILLI BABU	35	M	1	2	1	2	2	1	1	2	1	2	2	1	2	2	2	2	2	2	M	2	1	2	2	2	2	2	1	2
279	MUNIAMMA	65	F	2	2	2	2	2	2	1	2	2	2	1	1	1	2	2	2	2	1	M	1	2	2	2	2	2	2	2	2
280	CHRISTY	33	M	1	2	1	2	2	1	1	2	2	2	2	1	2	2	2	2	2	25	D	2	1	2	2	2	2	2	2	2
281	RAMESH	31	M	1	2	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	M	2	2	2	1	2	2	1	2	2